The New England
Journal of Medicine

VOLUME 209 OCTOBER 19, 1933 NUMBER 16

Massachusetts Medical Society

SECTION OF TUBERCULOSIS

Monday, June 5, 1933

PRESIDING:
Dr. Roy Morgan, Westfield, Chairman.
Dr. Donald S. King, Boston, Secretary.

CHAIRMAN MORGAN: The meeting will come to order, please. First on the program is the appointment of the Committee to present nominations for officers for the ensuing year. For that Committee I would like to appoint Dr. Albert C. Getchell, Dr. Sumner H. Remick, and Dr. Moses J. Stone.

Is there any other business to come before the Section?

DR. FREDERICK T. LORD, Boston: *Mr. Chairman*—We have lost by death an esteemed and prominent member of this organization, Dr. Edward O. Otis. I should like to move the appointment by the Chair of a committee to draw up a resolution with respect to the death of Dr. Otis, that this resolution may be spread upon our records and a copy sent to his family.

(The motion was seconded and carried.)

THE CHAIRMAN: For that committee I would like to appoint Dr. Frederick T. Lord, Doctor Churchill, and Doctor King.

Is there anything else to come before the Section? If not, the first paper on the program is "Pulmonary Tuberculosis in Adolescents with Special Reference to Frequency, Diagnosis and Prognosis", by Dr. Alton S. Pope, of Newtonville.

PULMONARY TUBERCULOSIS IN ADOLESCENTS WITH SPECIAL REFERENCE TO FREQUENCY, DIAGNOSIS, AND PROGNOSIS*

BY ALTON S. POPE, M.D.†

BETWEEN the ages of three and fifteen tuberculosis takes a lower toll than at any other period of life, yet it is just at this time that the greater part of tuberculous infection takes place. Careful studies of autopsy data on patients dying from various causes indicate that by the age of 20 nearly everyone shows some evidence of tubercle. More recently widespread application of the tuberculin test has made it possible to get a fairer cross section of the population, and to estimate the extent of infection at different age periods. Showing, as it does, individuals at some time infected with tubercle bacilli the tuberculin test indicates much more accurately than the incidence of reported cases or deaths, the actual frequency of tuberculous infection in the community. Roughly speaking, the proportion of children who react to the test rises steadily from birth to about 20 years. The number infected at a given age has been found to vary widely in different parts of the country, or even in different sections of the same city. Recent figures[1] for white children of 15 have shown a range from 12 per cent of reactors in rural Minnesota to as high as 77 per cent in Philadelphia.

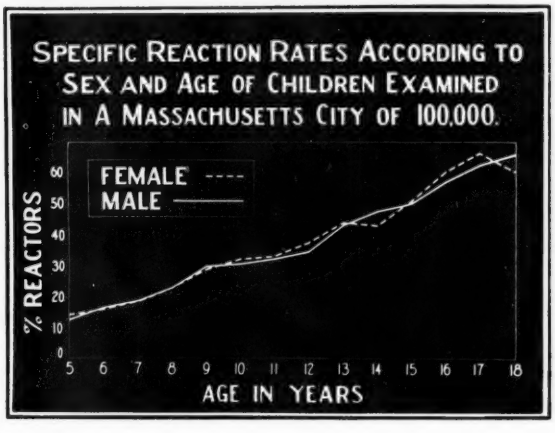

FIGURE I. Percentage of children reacting to the tuberculin test, according to age and sex, 13,582 examinations in a Massachusetts city.

It has long been recognized[2] that the tuberculosis of childhood differs markedly from adult phthisis and that it is essentially a primary infection, benign in character, but a necessary an-

*Read before the Section of Tuberculosis of the Massachusetts Medical Society at Boston, June 5, 1933.

†Pope—Director, Division of Tuberculosis, Massachusetts Department of Public Health. For record and address of author see "This Week's Issue," page 811.

tecedent to the progressive, fatal tuberculosis of adults. It is not within the scope of this paper to consider whether the adult type of tuberculosis is a direct extension of the childhood disease or, as many contend, the result of strictly exogenous reinfection. It is, however, with this distinction between the childhood and adult types in mind that we wish to discuss phthisis in children from the standpoint of frequency, diagnosis, and prognosis.

The data here presented have been drawn from three sources: the official mortality statistics of Massachusetts, case records of tuberculous children diagnosed and followed by the State School Clinics, and over 20 years' experience in the hospitalization of tuberculous children at the Westfield State Sanatorium.

FREQUENCY

Among Massachusetts school children the rate of reaction to the tuberculin test has been found to rise quite regularly from about ten to fifteen per cent at five years of age to about 45 to 55 per cent at the end of high school life. At no time is there any substantial difference in the reaction rates in boys and girls.

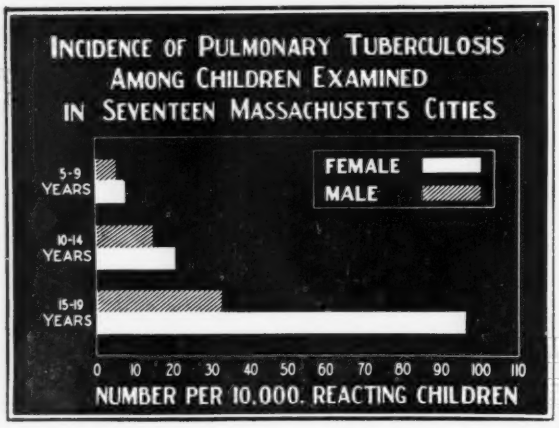

FIGURE II. Proportion of children who develop pulmonary tuberculosis per 10,000 reactors, according to age and sex—examinations in 17 Massachusetts cities.

In contrast to this equal rate of infection in males and females up to the age of eighteen, Figure II shows that above the age of nine, pulmonary tuberculosis is found in an increasingly greater proportion of the infected girls than of the infected boys.[3] In the ten to fourteen year group the incidence of pulmonary infiltrations is one and one-half times as high, and in the fifteen to nineteen group three times as high in girls as in boys of the same age. This difference in the incidence of pulmonary tuberculosis in males and females is consistent with the difference in the tuberculosis death rates for the sexes at this age, and corresponds quite closely to the ratio between the numbers of boys and girls who are admitted to our sanatoria. From our data it would appear that girls at this age period tend to develop pulmonary tuberculosis approximately three years earlier than boys.

Since the rates of infection in males and females are essentially the same at this age, it is unlikely that girls receive more frequent or massive doses of tubercle bacilli than boys. There is no reason to believe that there can be any difference in the virulence of the organisms involved in the two sexes. Hence, the only logical explanation of the difference seems to be a greater susceptibility to the development of pulmonary tuberculosis among infected females at this age period.

In a group of 100,000 school children examined in the above-mentioned seventeen Massachusetts cities, the morbidity rate in the ten to fourteen year group was eighteen, and in the fifteen to nineteen group sixty-five per 10,000. If these rates may be taken as representative of the State as a whole it would indicate at least 3000 cases of pulmonary tuberculosis, largely unrecognized, in Massachusetts children between the ages of ten and nineteen.

Although the death rate from tuberculosis is relatively low in the age group ten to nineteen, the disease in Massachusetts is responsible for more than three times as many deaths as all of the acute communicable diseases together at that period of life. Furthermore, the deaths in adolescent children represent only a fraction of the actual damage which results from tuberculosis at that age. In our Follow-Up Clinic Zacks[4] has found an average interval of some three

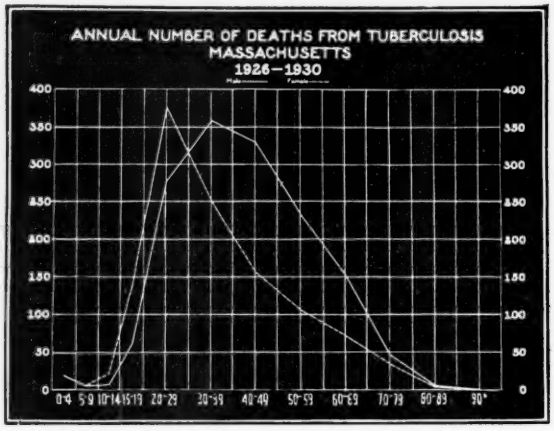

FIGURE III. Age distribution of deaths from pulmonary tuberculosis in Massachusetts, according to sex.

years between the x-ray diagnosis of pulmonary tuberculosis and the development of clinical symptoms. Add to that the two or three years that usually elapse between the first symptoms and the time of death, and it becomes evident that a great part of the maximum tuberculosis mortality, from twenty to twenty-four, has its beginning during adolescence. This relationship is readily visualized from the accompanying figure on the age distribution of tuberculosis deaths.

The tuberculosis death rate of children in this group has declined approximately eighty per cent since the first of the century, a gain slightly greater than that in the tuberculosis rate for all ages. It will also be noted that the rate of

decline has been more rapid for the past ten years than ever before. This trend is interesting in view of many loose statements which have been made concerning the effects of the present fashionable inadequacy of diet and clothing upon the tuberculosis death rate in our girls and young women. The situation here is in marked contrast to the corresponding age groups in England and Wales where during the same period the tuberculosis death rate has shown only a forty per cent decrease among girls from ten to fourteen, and no decline at ages fifteen to nineteen.

It is at this age that the excess mortality from tuberculosis in females becomes evident, an excess which continues up to the age of thirty-five. It will, however, be seen in the diagram below that while the incidence of tuberculosis among females is twice as high as in males, the rate of decline has been approximately the same for both.

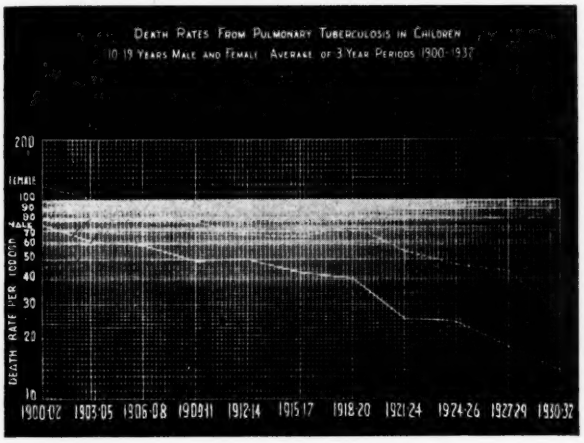

FIGURE IV. (Slide, semi-log.) Death rates from pulmonary tuberculosis in children 10-19 years, male and female, by 3 year periods—1900-1932.

DIAGNOSIS

The most serious problem in tuberculosis of children of this age is to establish the diagnosis while the patient still has a fair chance of recovery. Often children with moderately advanced to far advanced lesions show no loss of weight or strength, and no symptoms to suggest the very grave prognosis of their disease. Of 130 pulmonary cases found in the Chadwick Clinics from 1924-1929 only eight per cent gave definite symptoms, and barely fifty per cent showed either symptoms or physical signs. Without the routine tuberculin test and x-ray examination of the reactors it is obvious that practically none of these cases would have been discovered until the disease was much farther advanced.

At this age the diagnosis of early pulmonary tuberculosis is almost wholly dependent upon competent roentgenological examination. All soft, flocculent, apical or sub-apical densities must be regarded with suspicion, and if they persist beyond the time in which a transient pneumonic process can be expected to clear, the only safe procedure is immediate and intensive treatment. In practically all cases this means hospitalization. Waiting to see whether the process will take care of itself is simply throwing away the patient's chance for recovery in many of these cases. To rule out transient infiltrations, a second chest plate should regularly be required in making a diagnosis of pulmonary tuberculosis in children. Symptoms and physical signs if present are of definite confirmatory value, but their absence must not be taken to rule out tuberculosis. In fact, in our school clinics the majority of children who presented pulmonary symptoms or signs proved to be suffering from non-tuberculous conditions. Slight persistent cough, fatigue, and failure to gain weight, may be associated with early tuberculous processes, but tend to appear when the disease is more advanced. Only a fifth of our cases had râles at the time of diagnosis.

In a careful study of 1000 high school students in New York[5] recently, employing the tuberculin test, x-ray, and physical examination, fifteen children were found to have manifest tuberculous disease. In none of these could the process be demonstrated by symptoms or physical signs, while among the twenty-four in whom suspicious physical signs were detected none proved to be suffering from tuberculosis.

Exposure to pulmonary tuberculosis in the household is by far the most important item in the history of these children. Among children living in contact with known cases the rate of reaction to the tuberculin test is from two to four times[6] as high as in other children, according to age. In a series of pulmonary cases followed from three to seven years Zacks[7] was able to find definite contact with pulmonary tuberculosis in the family in sixty-five per cent. The tuberculin test is valuable not only in reducing the number of children to be x-rayed, as in school examinations, but is also helpful in differentiating tuberculous and non-tuberculous processes in certain instances.

Objection is often raised against making a diagnosis of tuberculosis on x-ray evidence alone, and requiring sanatorium treatment for children who are apparently in good health. The only answer is our experience with this type of case. The children who were hospitalized for pulmonary tuberculosis before the days of x-ray diagnosis practically all died. It is true that a certain number of minimal apical infiltrations in children remain stationary for periods of one to three or four years. A few clear without ever giving rise to active disease. The great majority if untreated spread insidiously year by year until they flare up as manifest tuberculosis. Since it is not possible to determine

the activity of such lesions by a single x-ray plate, the only safe rule is to recommend sanatorium care with complete bed rest if the infiltration is soft and flocculent in character[8]. Doubtful processes should be carefully followed with x-ray examinations at least once a year and the patient promptly hospitalized as soon as any roentgenological evidence of spread appears. The danger of making treatment wait upon clinical confirmation of the diagnosis is illustrated by the first case shown in our slides.

PROGNOSIS

Because pulmonary tuberculosis in adolescents often remains clinically "latent" for periods of several years, there has been a tendency to minimize the gravity of its ultimate prognosis. It is only when a group of tuberculous children are followed for a considerable time that the true fatality of the disease appears. The practice of reëxamining annually all children not hospitalized, in whom a diagnosis of any form of tuberculosis has been made in the Chadwick Clinics, has now provided a group in which results can be traced for a period of four to eight years.

Out of a total of 88 school children in whom a diagnosis of pulmonary tuberculosis was made during the period 1924 to 1929, twenty-five, or twenty-eight per cent, are now known to be dead; seven more were unimproved at the last examination, and twenty-four could not be traced. Only nine of the group appeared to be entirely well. As this group includes all pulmonary cases diagnosed in five years' work of the clinic, whether hospitalized or not, it is the best index we have of the true fatality of pulmonary tuberculosis at this age.

Since its establishment in 1910 the Westfield State Sanatorium has admitted children with both adult and childhood types of tuberculosis. Altogether, over 600 cases of pulmonary tuberculosis in children have been treated at the institution. During the summer of 1932 an attempt was made to trace all children formerly treated at the Sanatorium for the adult type of disease, to secure if possible some measure of the efficacy of treatment and at the same time determine the actual fatality to date.

The ages of these children ranged from ten to 17 years at the time of admission. For convenience they have been divided into two periods, from 1910 to 1920, and from 1920 to 1927. As there was no x-ray apparatus available at the institution until 1920 the first group was limited to children who had a positive sputum at some time during their illness. In the 1920 to 1927 group, diagnosis depended upon x-ray findings and the tuberculin test, with or without confirmatory clinical evidence. In the first series rest treatment was not carried out in the present form. Patients were kept in bed only when they had fever, and were allowed up within a few days after the temperature became normal. None of them received collapse therapy of any kind.

TABLE I*

PRESENT CONDITION OF CHILDREN WITH PULMONARY TUBERCULOSIS ADMITTED TO THE WESTFIELD STATE SANATORIUM

March 1, 1910—February 29, 1920

Stage on Admission	Number of Admissions	Not Traced	Living	Dead	Per Cent Dead
Minimal	12	5	2	5	42%
Moderately Advanced	44	12	1	31	71%
Advanced	144	16	4	124	86%
Totals	200	33	7	160	80%

*Data from Westfield State Sanatorium, by courtesy of Roy Morgan, M.D., Supt.

TABLE II*

PRESENT CONDITION OF CHILDREN WITH PULMONARY TUBERCULOSIS ADMITTED TO THE WESTFIELD STATE SANATORIUM

March 1, 1920—February 28, 1927

Stage on Admission	Number of Admissions	Not Traced	Living	Dead	Per Cent Dead
Minimal	22	5	8	9	41%
Moderately Advanced	61	9	22	30	49%
Advanced	112	10	8	94	84%
Totals	195	24	38	133	68%

*Data from Westfield State Sanatorium, by courtesy of Roy Morgan, M.D., Supt.

On account of the advanced stage of disease on admission little could be expected from treatment in the first group of patients. It is disturbing to find so high a fatality among minimal cases in the second series, but even at that time bed rest was much less strictly carried out than at present, and pneumothorax was used in only a few cases. With more intensive treatment the results in patients admitted from 1927 to 1929 appear more promising, but several years must elapse before definite conclusions are warranted.

CASE HISTORIES

CASE I

I. F., female, aged 12.

Examined in school, March, 1929; ten per cent underweight, but in good health; only complaint was cough of four or five days' duration. No history of contact was obtained. Patient reacted to the tuberculin test, and on x-ray findings was diagnosed as a "suspect" case.

The following year patient's condition was unimproved. At that time her father was found to have pulmonary tuberculosis.

In 1931 there was definite mottling at the right apex with râles, slight cough and occasional expectoration, and a diagnosis of pulmonary tuberculosis was made. Sanatorium treatment was refused on advice of the family physician.

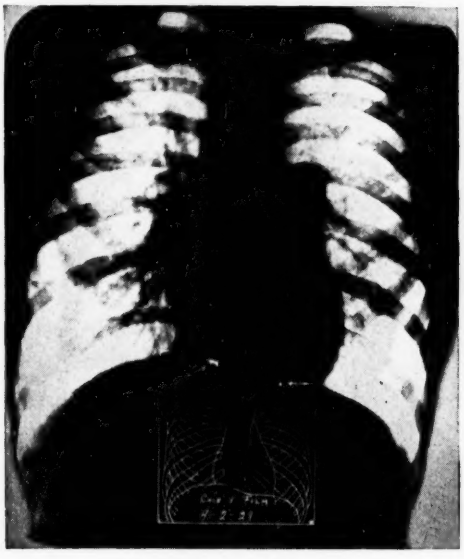

FIGURE I, CASE I.

Examination in 1932 showed extension of infiltrative process at the right apex, with fine crepitant râles. Sanatorium treatment was again urged, but refused on advice of a consultant that it was unnecessary. Patient denied symptoms.

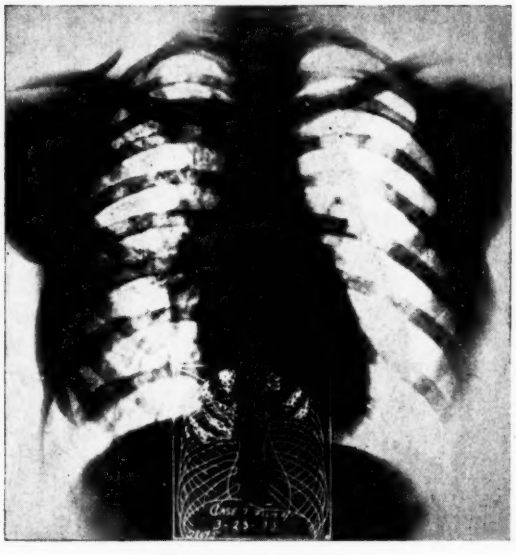

FIGURE II, CASE I.

In 1933 there was marked extension of the pulmonary process with involvement of most of the right lung and of left apex—pulmonary tuberculosis, stage 3. Patient still looks well and denies symptoms.

This case illustrates very well the steady, insidious progress of tuberculosis, characteristic of children at this age. Without routine x-ray examination the majority of such cases are beyond help when the diagnosis is made. Further delay in treatment takes away what chance remains.

CASE II

P. J., male, aged 10.

Examined in school, December, 1924; normal weight, very active, no symptoms. Contact denied.

FIGURE III, CASE II.

Classified as latent pulmonary tuberculosis; was reexamined annually. In June, 1926 was said to have been ill with pleurisy. Admitted to the Westfield State Sanatorium December 18, 1926 as an advanced case of pulmonary tuberculosis. He was discharged apparently arrested on November 29, 1930. In June, 1931 he was reëxamined at the Outpatient Department at Westfield, with the condition shown in the accompanying film. Shortly afterwards he was admitted to the Springfield Tuberculosis Hospital with far advanced pulmonary tuberculosis. He remained there less than a year, and died at home in July, 1932.

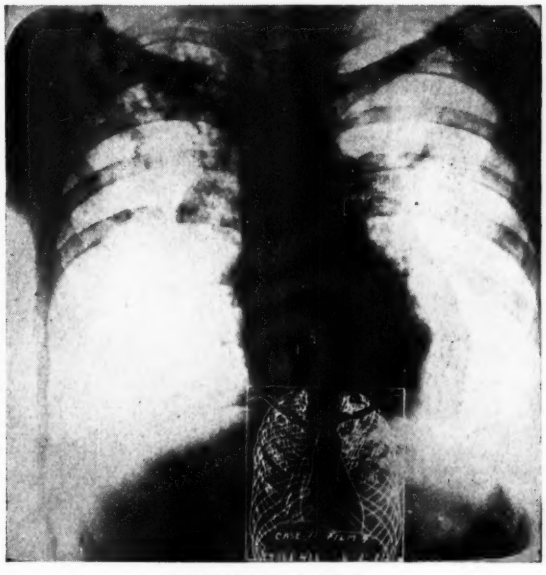

FIGURE IV, CASE II.

Comment on the danger of considering apical pulmonary infiltrations at this age as quiescent seems superfluous. This case also shows the very deceptive course of phthisis in children, and

the relatively long periods of treatment required before a process can safely be considered arrested.

CASE III

L. D., female, aged 14.

Examined in school, May, 1925; twenty-five per cent underweight, but in good health, very active, no cough or other symptoms. Her mother had died of pulmonary tuberculosis. Examination showed dullness at the top of the right lung, front and back, with fairly numerous fine râles. X-ray examination revealed the extensive lesion shown, and a diagnosis of pulmonary tuberculosis was made. Admitted to the Westfield State Sanatorium on June 30, 1925, stage, moderately advanced; transferred to the North Reading State Sanatorium as improved April 12, 1926; discharged from North Reading as quiescent July 2, 1927. Last report was that she is living in Texas, in good health and married.

Cases of this type indicate that prompt and long-continued treatment is effective in a certain proportion of cases. Our experience has been that prognosis depends chiefly upon the discovery of pulmonary infiltration at the earliest recognizable stage. Even patients with apparently arrested process frequently break down under the strain of child-bearing or work.

CASE IV

E. J., female, aged 15.

First seen in March, 1930; in school, slightly overweight, with no symptoms. Physical findings were normal except for harsh breath sounds and slight dullness at apices. At that time a diagnosis of minimal pulmonary tuberculosis was made on x-ray evi-

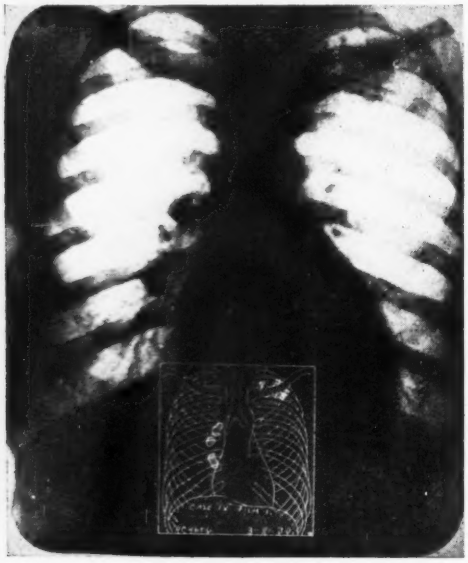

FIGURE V, CASE IV.

dence. Admitted to the Westfield State Sanatorium July 18, 1930. Patient did well until May 7, 1931, when she began running a temperature from 100° to 104°. From that time the lesion progressed rapidly, and the patient died July 27, 1932.

Pneumothorax was advised June, 1931, but the family refused consent until the process had spread to the other lung.

The explosive spread of the pulmonary lesion this patient experienced while apparently responding favorably to treatment is not uncommon at this age. Such cases almost invariably do badly in spite of any form of treatment.

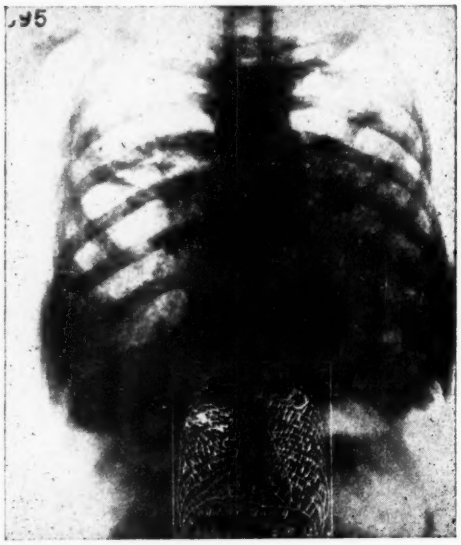

FIGURE VI, CASE IV.

CASE V

C. V., female, aged 15.

Examined in school, May, 1925; ten per cent underweight, no symptoms. History of frequent colds; exposure to cousin in family five years. Dullness right top, with harsh breath sounds, no râles heard. Diagnosis of pulmonary tuberculosis on x-ray.

Admitted to the Westfield State Sanatorium July 25, 1925, classified as moderately advanced; transferred to the North Reading State Sanatorium, condition improved, April 12, 1926; transferred to the Rutland State Sanatorium, condition unimproved, August 29, 1930; died at the Rutland State Sanatorium February 11, 1932, after nearly seven years of hospital treatment.

Again we find an apparently well child in school with a pulmonary process so advanced that long and expensive hospitalization served only to postpone the issue.

SUMMARY

1. In Massachusetts between the ages of ten and nineteen pulmonary tuberculosis still takes a toll of more than three times as many deaths as all of the acute communicable diseases combined.

2. In view of an average duration of five years from the first x-ray signs of pulmonary infiltration to death, it is evident that a great part of the high tuberculosis mortality of the succeeding decade is a direct result of the unrecognized incipient disease of adolescence.

3. Because pulmonary tuberculosis in adolescents rarely gives sufficient symptoms and signs to warrant a diagnosis until the process is relatively advanced, the only practical method of discovery is x-ray examination of children during this age period.

4. Waiting for the development of symptoms and signs before starting treatment in a child of this age who shows tuberculous infiltration is simply throwing away the patient's one real chance for recovery.

5. The prognosis of pulmonary tuberculosis in adolescent children is grave, even when discovered early. Patients at that age respond less certainly to treatment, require longer periods to secure an arrest, and are more subject to relapse than adults.

6. In a group of 88 school children in whom a diagnosis of pulmonary tuberculosis was established four to eight years ago, there has been a subsequent mortality of twenty-eight per cent.

7. In two groups of children with pulmonary tuberculosis hospitalized between 1910-1920, and 1920-1927, there have been, respectively, fatalities of 80 and 68 per cent.

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Discussion

Dr. F. T. Lord: I welcome this opportunity to comment on Dr. Pope's important contribution. The investigation of tuberculosis in children on a large scale by the State Department of Public Health during the past nine years has offered the opportunity to accumulate evidence not otherwise obtainable. The data presented by Dr. Pope modify the conception of the adult type of the disease in children.

We know for the first time the magnitude of the problem with respect to the adult type of tuberculosis in children in Massachusetts. We now know that there are at least 3000 such cases in the State and it is fair to assume that a large proportion are unsuspected and uncared for victims of the disease.

With respect to the recognition of the disease, an important contribution has been made. It is now known that at the inception of the pulmonary type in children there is a long, latent interval, averaging three years, during which the diagnosis can be made only by a positive skin test with tuberculin and an x-ray examination of the reactors. It is fortunate with respect to the diagnosis by such means that the presence of fine or coarse mottled increase of density above the anterior portion of the third rib is rarely due to other causes than tuberculosis. Nontuberculous bronchopneumonia may usually be differentiated by a more stormy onset, leucocytosis and evidence by x-ray of involvement of other parts of the lung than the apical region. In doubtful cases, the course of the disease will help in the distinction. With nontuberculous pneumonias, the fever terminates in favorable cases usually between the seventh and the tenth days, with rapid subsidence of the increase of density, while tuberculous processes subside only very slowly or not at all.

Important evidence has been accumulated regarding the appalling gravity of the adult type of tuberculosis in children. The group of 88 children derived from the Chadwick Clinics with a mortality of 28 per cent and only nine entirely well after four to eight years is most significant with respect to the evolution of the disease.

In the prevention and control of tuberculosis the finding of the disease in a child is only a first step and should be followed by the examination of all household contacts by skin tests with tuberculin and clinical and roentgen-ray studies. By this means the discovery of tuberculosis, often in an early and favorable stage, may be expected in about one in four or five other members of the patient's family. Facilities for roentgen-ray examination are available on request by physicians through the state and county sanitaria, by extension of their service to include neighboring localities not otherwise covered and by many municipalities.

The serious outlook with pulmonary tuberculosis of the adult type in children makes it desirable to institute treatment at once. Lack of symptoms is likely to lead to a false sense of security and consequent delay during which the disease is likely to advance to a stage from which an arrest is not to be expected. With the discovery of pulmonary tuberculosis in a patient under 20 years of age, therefore, it seems imperative to advise immediate, long-continued, absolute bed rest even in the absence of indications of activity of the disease. The length of time the bed rest should continue is difficult to estimate and must depend on the progress under observation and especially the result of x-ray examination. In patients in whom the progress is satisfactory the rest treatment had best be continued until a period of many months has elapsed without symptoms and, more important still, until there is evidence by x-ray of disappearance of the lesions or an arrest of the disease by fibrosis or calcification. Lack of favorable progress under treatment by bed rest makes it desirable to resort to collapse therapy in selected cases.

The Chairman: This paper is now open for general discussion. Will any one wishing to discuss the paper please announce his name?

DR. S. A. MAHONEY, Holyoke: The subject of the paper is "Pulmonary Tuberculosis in Adolescents with Special Reference to Frequency, Diagnosis and Prognosis." Now I should very much like to take up the last subdivision, the prognosis. Some ten or fifteen years ago the State Department of Health of Massachusetts compelled every municipality above 50,000 to build sanatoria for communicable diseases, both contagious and tuberculous. In our city we had to expend over $50,000 in erecting buildings in order to take care of these cases. We had a survey of our health conditions by a professor from the Institute of Technology, about four or five years ago, and he advocated the entire elimination of about every building that the State Department of Health four or five years previously had compelled—not advised but compelled—the city to erect.

Now when you attempt to improve the prognosis in adult and adolescent tuberculosis, the surroundings and care of the patient are the main determining factor. We have these municipal sanatoria. Now it is a question whether the State Department of Health is exercising the proper function over these municipal sanatoria in order to better the prognosis and diagnosis. They can compel a municipality to put up those sanatoria and then allow them to develop their activities, according to the desires of the politicians of the city. I would like to know if it is not within the power of the State Department of Health to see that proper officials are put over these sanatoria and proper treatment given to adult and adolescent tuberculosis patients in the primary, secondary and terminal stages of the disease. In our sanatorium, which I have lately visited, there were thirty-five cases with only four or five bedridden, and about thirty ambulatory. Not one of them had any of the advances in treatment of tuberculosis such as pneumothorax (collapse therapy) or anything else, nor any attempt at such a thing. When we get to the prognosis wouldn't it be well, instead of trying the Chadwick Clinic fad and other things of that sort, to see if we cannot arrest the disease after it has started and see if the State Department of Health has not the power to compel a municipality to put in officials who will see that the patients in those buildings have proper treatment thereby improving the prognosis? I thank you.

THE CHAIRMAN: Does anyone else care to discuss the paper?

DR. CARL R. O'BRIEN, Bangor, Maine, formerly of Boston: I am interested in children and in childhood tuberculosis. Although the President sets apart a week for recognition of childhood tuberculosis and states set apart days or weeks, there is a trend on the part of municipal sanatoria today to get rid of advanced cases of tuberculosis to make room for those more favorable or more amenable to treatment. Now heredity we recognize as social rather than biological. What is the source of our childhood tuberculosis but the far advanced cases going around and spitting millions of organisms capable of entering the body and causing disease? It seems to me you are defeating your own ends when you are spending a lot of effort and money on the treatment of tuberculosis in children and sending out your uncontrolled advanced cases to wander around the streets and be a source of childhood tuberculosis.

SPEAKER FROM THE FLOOR: I was particularly interested in this paper of Dr. Pope's as showing the value of x-ray in early diagnosis. Back in 1908, 1910 and along there I was on duty as an x-ray man at the Boston Dispensary with the late Dr. Granger and the late Dr. Frederick W. Bardwell, and my attention was early attracted to the value of the x-ray in diagnosis for tuberculosis. Dr. Gibson of Denver, Colorado, had been reading papers concerning the cures of tuberculosis by x-ray, but he did not call any special attention to its value in diagnosis. It occurred to me that if it had any value in the curative field, it certainly must have value in diagnosis. I went to Dr. Otis, who was then in charge of the tuberculosis clinic, and asked him to send us some cases. At that time there had been practically little work done, so far as I know, in x-ray diagnosis of tuberculosis, but Dr. Otis became interested. In the beginning he was a bit hesitant, but it was not long before he was having all of his new cases x-rayed.

In 1924 I was on duty in the Veterans' Tuberculosis Hospital in New Haven and I was particularly interested and gratified to find at that time that the Director of the Veterans' Bureau had issued the statement that the x-ray was perhaps the most important diagnostic agent we have for tuberculosis in its early stages. This paper of Dr. Pope's has abundantly demonstrated that possibility.

If there is anything that is more pathetic or more disheartening to a doctor than to be on duty in a tuberculosis hospital, as I was, and see sometimes two or three who should have been under ordinary circumstances fine, robust young men, dying and being taken away by the undertaker, I don't know what it can be. And anything that will offer hope of early diagnosis and possible elimination of this terrible disease in the individual is certainly worthy of our greatest support.

DR. JOSEPH PRENN, Boston: I was called in to examine the throat of a girl, of about 18. There was a history of hemoptysis with repeated negative sputum and negative physical chest signs.

The crypts of the tonsils were filled with secretions that looked like sago and gave a picture resembling pharyngomycosis. Believing, however, that they might be tubercular, I advised their removal. As it would have to be done under local anesthesia the patient objected

at the time. It could now be done, I suppose, under avertin or with the electrosurgical snare and knife.

I followed the case for about a year and a half, cleaning out the crypts and giving her local antiseptic applications. During this time the tonsillar structure was falling apart. There was glandular destruction. Whenever I thought she was fit for the operation she thought herself well enough not to need one, and whenever she wanted it, I did not think she was fit for the operation. When finally I removed the tonsils there appeared three glistening foci on the capsular side of the tonsil.

The report from the laboratory was miliary tuberculosis of the tonsil. This case brings out the following facts:

1. While glandular destruction was going on anteriorly in the tonsillar substance, the foci of infection, caseation and liquefaction were taking place in the capsular side of the tonsils where the blood supply is, and the vessels enter and leave the tonsils.
2. The tonsils are capable of being infected with true tuberculosis, unlike other glands of the body which are supposed to harbor the bovine type.
3. The danger of procrastination.

THE CHAIRMAN: Anyone else? Dr. Pope, will you close the discussion?

DR. POPE: I have nothing to add, except that I would like to express my appreciation of Dr. Lord's bringing out the fact that the State Department of Health is trying to offer physicians diagnostic service for the patients who cannot afford to go to private roentgenologists. It seems to me, since perhaps x-ray is the most essential single service that we have, that we will get farther in finding early tuberculosis if we make the x-ray service available to patients referred from anywhere in the State than we will by any other procedure. Such service to physicians is now being offered at the State and County Sanatoria, and at about fifteen diagnostic clinics which are staffed by men from these same Sanatoria.

In regard to Dr. Mahoney's question, you all have heard the familiar aphorism that anybody can lead a horse to water but not many people can make him drink. I think his is largely a local problem. While the Massachusetts statute passed about fifteen years ago required cities of 50,000 and over to build tuberculosis sanatoria, it gave the State Department of Health no authority over such local sanatoria aside from approving the members of the staff and the condition of the hospital. In fact, such approval is necessary only for the payment of State subsidy and if the subsidy is withdrawn for failure to meet requirements the State Department has no further recourse in the matter. There is no way in which the State Department can tell any local department what it shall do and how it shall do it. There has been a striking improvement in a number of local and county sanatoria in the last two or three years. I do not see any legal way in which the State can actually force a hospital to make improvements if the hospital staff is unwilling to make them.

In regard to Dr. O'Brien's question, it seems to me that the most important measure, perhaps, is to minimize the spread from these sputum-positive cases that are roaming the countryside, first, by recognition of the cases. Many of them, who have never had a diagnosis of tuberculosis, are, as we know, now at large with positive sputum. Others who are considered quiescent may have had a reactivation of their process and become unconscious spreaders of the disease in their families. Through the routine examination of family contacts I believe we can locate more of these spreaders than in any other way. When these chronic infectious cases are located it is the job of the physician and the local board of health to teach and persuade them how to avoid infecting the members of their households and the community. Certain states have passed legislation authorizing forcible restraint of such known positive cases. I happen to have worked in a large city where such a law was enforced. In a population of over three million, about two cases per month were forcibly hospitalized, and many of these were the same patients hospitalized over and over again. If you force a patient into a hospital he can soon get out on habeas corpus. Furthermore, for every spreader of tuberculosis who is forced into a hospital there are probably at least ten other patients driven away from their private physicians or local clinics and so deprived of diagnosis and medical supervision. While it is extremely annoying to have these cases spreading infection around the community and not be able to do anything about it, I doubt very much whether compulsory hospitalization would get as far in the long run as offering medical service to all these people.

DR. MAHONEY: *Mr. Chairman*—May I say one word in regard to the last statement of Dr. Pope concerning the State Department of Health. I understand it has supervision over these local sanatoria. He says that the State Board has to approve of the men that are officers in each sanatorium. Then why haven't they secured control so that they can insist that proper men are there to do the work that has come out in tuberculosis in the past ten years, such as pneumothorax, phrenicectomy and the various other surgical procedures? If they have the supervision why do they not exercise it?

CHAIRMAN MORGAN: The next paper on our program is on "Some Conditions in which the Roentgen Examination May Lead to an Erroneous Diagnosis of Pulmonary Tuberculosis," by Dr. George W. Holmes.

SOME CONDITIONS IN WHICH THE ROENTGEN EXAMINATION MAY LEAD TO AN ERRONEOUS DIAGNOSIS OF PULMONARY TUBERCULOSIS*

BY GEORGE W. HOLMES, M.D.†

THE importance of the roentgen examination in the diagnosis of pulmonary tuberculosis is now generally accepted. No less an authority than Lawrason Brown of Saranac has placed it next to the demonstration of tubercle bacillus in the sputum as the most valuable single finding. It is also generally recognized that pathologic changes in the lung may elude the most painstaking physical examination, and that the roentgen examination is an essential part of a positive or negative diagnosis in pulmonary tuberculosis. This general acceptance of the importance of the roentgen examination is a matter of rather recent origin and is due in part to the vast amount of educational work which has been done by the leaders in this field of diagnosis, and in part to the great improvement in roentgen technique.

Today, the pendulum seems to have swung in the opposite direction. In some localities, at least, the interpretation of the roentgen findings is being accepted as final, and the necessity of checking these findings with the history and physical examination is neglected. For these reasons it seems appropriate at this time to discuss some of the conditions in which the roentgen examination, taken alone, may lead to an erroneous diagnosis of pulmonary tuberculosis.

The roentgen method of examination is essentially a laboratory procedure and is subject to all the errors to which this method of examination is liable, including mistakes in identity. The value of the findings will depend upon the care and skill with which the examination is made, and the training and experience of the physician who interprets the findings just as in any other method of examination. In addition to these personal factors, there are certain conditions which so closely simulate active tuberculosis that it may be impossible to differentiate them and tuberculosis by the roentgen examination alone; fortunately, in many of these conditions the diagnosis is easy when the roentgen findings are compared with the history and physical findings. To illustrate these difficulties in the roentgen examination and the importance of comparing its findings with the history and physical examination, I have selected several cases including the following conditions: prominent pulmonary markings in the normal chest; anatomical variations in the lung; acute infections; chronic non-tubercular infections; changes in the lungs of cardiac origin; malignant disease of the lung; diseases due to the inhalation of dust; carcinoma of the bronchi; pulmonary collapse and healed tuberculosis.

CASE 1. E. M.—304050. *Roentgen Findings:* In the lower two-thirds of both lung fields and in the region of the right upper lobe, near the hilus, there are numerous circular areas of decreased density which resemble soap-bubbles. These areas are about 2 cm. in diameter, and do not contain fluid levels. The interlobar pleura is definitely thickened as is the pleura at the right apex and left costophrenic angle. There is diffuse thickening of the larger lung markings.

History and Physical Findings: This patient had a cough for thirty years with attacks of pleuritic pain in the chest, accompanied by blood-streaked sputum. No night sweats, no dyspnea, no orthopnea until after tonsillectomy seven years before admission when he developed fatigue, dyspnea and occasional acute attacks of orthopnea associated with an increase in the amount of sputum; no bacillus of tuberculosis found. Symptoms have been worse for the past two years; moist and musical râles at both bases and at the top on the right side; the fingers were clubbed. *Diagnosis:* Cystic disease of the lungs, and question of bronchiectasis of the right upper lobe.

CASE 2. O. P. D.—2904. *Roentgen Findings:* There is soft mottled dullness throughout the greater portion of the right upper lobe. The extreme apex does not appear to be involved. At the left lung root there is a small dense shadow which may be a calcified gland. There is also some thickening of the larger lung markings in the region of the left upper lobe. The heart shadow is not remarkable. The outline and position of the diaphragm are normal. The respiratory movements are somewhat limited; there is a definite process in the lung which could be of tubercular origin.

History and Present Illness: Duration of illness, twelve days, cold and sore throat with racking cough and pain over midsternum, slightly dull over right anterior chest, slightly prolonged breath sounds, no râles. The day following admission the patient was better, but the fever continued. Four days later crepitant râles, exaggerated by cough were heard above the right third rib. Sputum was negative for the tuberculosis on two occasions. A film taken one week after admission was negative. *Final Diagnosis:* Resolving pneumonia.

CASE 3. G. G. *Roentgen Findings:* The entire right lung field is dull; the dullness is most marked in the lower half of the chest and in the upper portion the shadow is of a mottled character. There are no cavities visible. The outline of the diaphragm is obscured. The heart and mediastinal contents are displaced to the right. From the film alone this could be a case of advanced fibroid tuberculosis with displacement of the heart and mediastinum toward the affected side.

History and Physical Findings: A healthy young man, aged nineteen, was operated upon for subacute appendicitis. He coughed considerably during recovery from the anesthesia, and on the following day, but otherwise his convalescence was normal

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Read before the Section of Tuberculosis of the Massachusetts Medical Society at its Annual Meeting, June 5, 1933.

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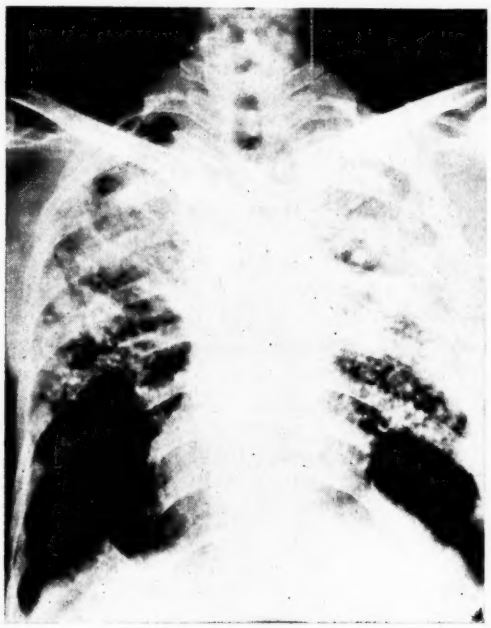
until the second day when he had a chill, with the onset of a cough, marked dyspnea and cyanosis. The respirations mounted to 48 and the temperature to 103°. Physical examination showed limitation of excursion of the right chest with eversion of the costal border. The right chest was dull throughout with diminished breath sounds, and in the region of the scapula was a patch of loud bronchial breathing, egophony, increased voice sounds, and tactile fremitus. No râles were present. The heart was displaced to the right. A moderate amount of purulent sputum showed pneumococci of type three. The respiratory embarrassment soon disappeared, the sputum became less and on the sixth postoperative day the temperature became normal and remained so. A film taken three days after operation showed a picture quite characteristic of massive collapse. The lung was rather slow in clearing up, and the changes had not entirely disappeared at the time of the patient's discharge, although he was practically symptom-free. This case is probably one of massive collapse.

CASE 4. Hos. 301611. *Roentgen Findings:* There is rather coarse mottled dullness throughout the upper half of both lung fields, the shadows extend into



CASE 4. There is rather coarse mottled dullness throughout the upper half of both lung fields, the shadows extend into the apices; the lower half of both lung fields are bright. The outline of the diaphragm is distinctly seen. The heart is not displaced. There are no visible cavities.

the apices; the lower half of both lung fields are bright. The outline of the diaphragm is distinctly seen. The heart is not displaced. There are no visible cavities.

History and Physical Findings: A forty year old man—a stone cutter for twenty-four years—entered the Hospital complaining of left pleuritic pain of nine months' duration with a slight cough productive of white sputum lasting for three to four weeks which has continued with dyspnea until the last few days. Weight has dropped fourteen pounds, and he has slight lassitude. Pain recurred two weeks ago with a slight increase in other symptoms; but in the past few days he has felt rather well and able to return to work. Physical examination showed poor teeth, chest contracted in front with flaring of C-M, retraction of the apices and breath sounds increased, particularly in the anterior lower lateral and posterior mid-chest. *Final Diagnosis:* Pneumoconiosis.

CASE 5. J. S. Hos. 141567. *Roentgen Findings:* The left lung field is smaller and less radiant than the right, and near the root of this lung there is a dense mass with indefinitely defined borders, beyond it in the lung field are areas of mottled dullness extending to the apex. The right lung appears normal. The heart is slightly displaced to the left.

History and Physical Findings: Patient, a fisherman, enters the Hospital with complaint of cough and pain in the left side of the chest. Six weeks before entering he "caught cold on board ship" and except for cough he felt well. He went to bed; his cough became worse and he had severe chills. He was removed to his home where his local physician found he had a temperature of 104°. His cough was paroxysmal in type, occurring chiefly in the morning. The amount of sputum raised at first was from two to three cupfuls a day. This gradually diminished. He had difficulty in sleeping on his right side. During the past week he has felt much better and has been able to rest lying on his right side. Three days ago he noticed blood-streaked sputum which has persisted to this time; he has lost considerable weight and strength; there have been no night sweats. Bronchoscopy done a week after entering showed stenosis of the left lower lobe, primary bronchus. He improved after bronchoscopy and returned home, and for four months did fairly well and gained weight. A week before re-entering his cough increased and sputum was blood-tinged. Bronchoscopy was again done and at this time the specimen removed proved to be carcinoma. *Final Diagnosis:* Carcinoma of the bronchus.

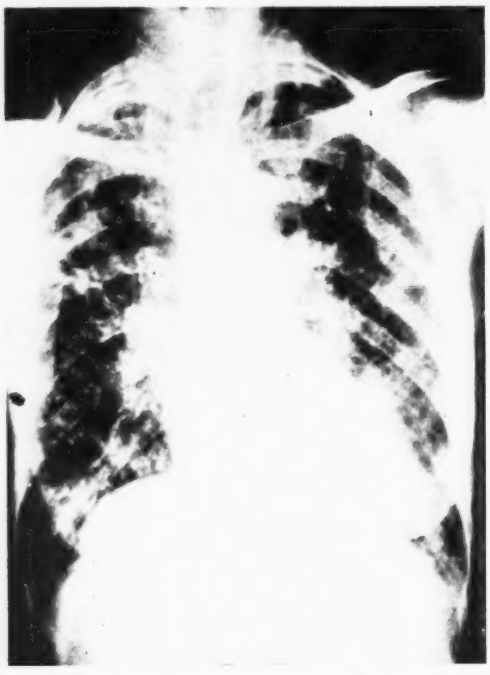
CASE 6. Hos. 115209. *Roentgen Findings:* The left lung field is smaller than the right, and there is mottled dullness which extends from the clavicle to the angle of the scapula, the changes are most marked around the root of the lung, and gradually fade out toward the periphery. The outline of the diaphragm is obscured on this side; the costophrenic sinus obliterated. The left hilus shadow is increased in size. The right lung field is clear, and unusually bright.

History and Physical Findings: A physician of fifty-four years enters for relief of pain in the chest. Pneumonia, seventeen years previously, recovery without incident; tonsillitis many times in childhood. He has been a morphine addict for thirty years. About two months ago he had an attack of acute bronchitis which lasted for two weeks. Three weeks before admission he experienced sudden dizziness and fainting spells; since this time has been bothered by pain over the left chest intensified by deep breathing and cough. He has also had dyspnea, marked loss of strength and a loss of about twenty pounds in weight. Physical examination showed a pale, emaciated man with pupils small, slightly irregular, reaction sluggish; teeth missing; chest, left side slightly more prominent than the right, expansion limited, breath sounds diminished over the left chest generally. Marked dullness in the left axillary line over the left front, and in the lower left back. The liver edge is about 4—5 cm. below the costal margin, it is irregular, hard and nontender. *Final Diagnosis:* Metastatic carcinoma of the lung.

CASE 7. Hos. 309482. *Roentgen Findings:* There is rather coarse mottled dullness, throughout the greater part of both lung fields, the changes are most marked in the right upper lobe, and around the root of the lung. The diaphragm is low, and there is evidently some emphysema present. The heart

is slightly enlarged to the left, there is no displacement, and there are no visible cavities. There is some retraction of the trachea toward the right. The appearance is that of a chronic process of a broncho-pneumonic character involving both lungs.

History and Physical Findings: A single, unemployed white woman, sixty-six years of age entered the Hospital on October 13, 1930 complaining of pain in the right side for six months. She dated her illness back to six years ago when she suddenly had an hemoptysis of 250 cc. Six months later she developed a severe cold and her local doctor told her she had lung trouble. She kept on working in spite of increasing weakness, colds and the raising of one to two ounces of white sputum daily. This continued until three years ago, when her local doctor prevailed upon her to enter a sanatorium where she has been for the past two and a half years. While in the sanatorium she had been raising four to eight ounces daily of white, thick, non-foul sputum, which had never been positive for tuberculosis. She weighed ninety pounds two years ago, and at present her weight is less. The right chest is hyperresonant. Excursion was limited; tactile fremitus was increased, breathing was vesicular. There were coarse crepitant, palpable and audible râles throughout. The whispered voice had a bronchial character. The left chest showed similar, but not so marked, signs as on the right. The sputum was negative for tuberculosis. *Final Diagnosis:* Chronic bronchitis with bronchiectasis; necropsy.



CASE 7. There is rather coarse mottled dullness, throughout the greater part of both lung fields, the changes are most marked in the right upper lobe, and around the root of the lung.

CASE 8. X. R. 171319. *Roentgen Findings:* There are hazy mottlings in the right infraclavicular region continuous with the hilus, but it does not reach the extreme periphery of the chest. The interlobar septum on the right side is faintly seen; there are no visible cavities. Except for the area described the lung fields are clear. The diaphragm appears normal. The findings are those of consolidation in the right infraclavicular region with evidence of interlobar pleurisy.

History and Physical Findings: Tonsillectomy two weeks ago followed by cough, slight yellow sputum and soreness in the left chest. Lungs negative. Heart, systolic murmur; temperature 100-102°; white blood count 11,000. Four days later there was dullness with harsh breath sounds and râles at the right apex; the breath was foul. *Final Diagnosis:* Lung abscess—healed.

CONCLUSIONS

From a review of this material it is obvious that pulmonary tuberculosis in any of its various manifestations may be closely simulated by other diseases of the lung, and it would seem to be a wise procedure on the part of the roentgenologist to confine his interpretation to a description of the gross pathologic findings, with the possible statement that such findings are consistent with tuberculosis, until after consultation with the clinician.

Except when tubercle bacilli have been found in the sputum a positive diagnosis of pulmonary tuberculosis should not be made until all of the data including the history, physical and x-ray findings have been evaluated.

DISCUSSION

DR. FREDERICK W. O'BRIEN, Boston: I am sure you have all found Dr. Holmes' paper, as I did, a most informative one. It is the sort of discussion that we ought to have more often so that we may keep our feet on the ground. Dr. Holmes, however, is far too modest when he talks of his inability to diagnose tuberculosis by x-ray. I am sure that in the run-of-the-mill case he has no difficulty. To me, the importance of his presentation lies in the fact that he has demonstrated that there are many processes besides tuberculosis that must be considered in any survey of the chest. When you understand the anatomy of the lungs and that all these various chest conditions which he has considered present similar x-ray densities secondary to infiltration, exudation and consolidation, it is not surprising that we have no pathognomonic signs. The situation recalls a remark of Chevalier Jackson, "Everything that wheezes is not asthma." You might say in regard to this paper that everything that shows mottling is not tuberculosis.

I hope you will not fail to listen to the paper on pneumoconiosis tomorrow because it will bring up this subject again. In the first slide that Dr. Holmes showed there was an increase in linear markings throughout the lung fields found in at least 50 per cent of all normal persons over 45 years of age and which only rarely is indicative of pathology but commonly signifies a physiologic old age change. In some types of pneumoconiosis one does not see any other sign of intrapulmonary change except in the character of the linear markings. This is true particularly of the asbestos group of which we are seeing more and more. Asbestosis is not usually associated with tuberculosis. However, silicosis in certain parts of this country is, and

for that reason it is of more interest to the tuberculosis expert. If you will remember, in Dr. Pope's chart showing the morbidity incidence and mortality rate of tuberculosis for those over 45, the curve began to drop abruptly at that age, yet that is the very time when it begins to advance among silicotics, particularly the granite workers. All types of pneumoconiosis may present the appearance of miliary infiltration difficult to distinguish from tuberculosis by x-ray. Many of the cases simulating tuberculosis that Dr. Holmes showed, one would only see in a large general hospital such as he is connected with, and yet it is most necessary that such manifestations of disease, even though rarely seen, should be called to our notice. His presentation brings out very forcibly the necessity for follow-up and autopsies on these unusual cases, and also, I think, brings into rather striking relief the fact that the roentgenologist should not be a technician but rather a consultant.

CHAIRMAN MORGAN: Any further discussion?

DR. CALVIN G. PAGE, Boston: *Mr. Chairman*—Speaking from the laboratory point of view, it is possible sometimes to cultivate fungi from the sputum in cases where no tubercle bacilli have been found. A yeast, a monilium, or perhaps a mould will prove to be the organism that is the basis of the lung trouble. These fungi grow well on special culture media, such as Czapek's sucrose nitrate agar, Sabouraud's glucose agar, and beer-wort agar. Fresh sputum spread on the surface of either of these media in a Petri dish, and incubated for several days at from 24 to 30 degrees centigrade, may yield a characteristic growth that will be a help in diagnosis, if it recurs on several examinations.

CHAIRMAN MORGAN: Anything else? Dr. Holmes, do you wish to say anything further?

DR. HOLMES: I do not.

CHAIRMAN MORGAN: Before we go on with our program perhaps the Nominating Committee is ready to report.

DR. GETCHELL: The Committee has brought forward the following names for the officers for the ensuing year: For Chairman, Dr. Roy Morgan; for secretary, Dr. Donald S. King.

CHAIRMAN MORGAN: The nomination of Dr. Morgan for chairman and Dr. King for secretary is presented. Any other nominations?

On motion, the candidates nominated were elected.

CHAIRMAN MORGAN: The next paper on our program is "The Changing Aspect of Tuberculosis Treatment," by Dr. J. Burns Amberson, Jr., New York.

THE CHANGING ASPECT OF TUBERCULOSIS TREATMENT*

BY J. BURNS AMBERSON, JR., M.D.†

THE attitude toward the treatment of tuberculosis has changed and is changing because our knowledge of the evolution of the disease and the ultimate fate of the patient has increased in recent years with unprecedented fullness.

Considering the pulmonary form of the disease, because it is this which most often seriously affects the adolescent and adult sections of the population and because tuberculous lesions in the lungs and their regional lymph nodes are the depots to which most similar lesions in other parts of the body owe their origin, some of the basic facts which influence present views may be summarized as follows:

In its incipiency the lesion of clinical importance is usually exudative and lobular pneumonic in character and limited to a portion of a single lobe, often well below the lung apex.

Promptly after its inception the lesion often undergoes rapid changes for better or worse. Central caseation and peripheral extension may appear within a few days or several weeks. Excavation may attain a diameter of two or three centimeters in two to four weeks. On the other hand, healing by resolution may start early and remove large inflammatory deposits. Complete resolution, however, is very infrequent and the central caseous residues then require many months for adequate fibrous encapsulation. After extensive resolution the caseous residues often are demonstrable only roentgenographically, and sometimes they are so small as to be indistinguishable even by this means. However, if adequate fibrous encapsulation does not follow, these caseous deposits may at any time be the origin for new exacerbations with or without cavity formation.

Once the first lesion has caseated, ulcerated, and excavated into a bronchus, further spread of the disease frequently occurs by the infectious discharge, coming into contact with healthy tissues, as it passes through the respiratory and alimentary canals. The most common sites of the secondary lesions, so caused, are in the same or the opposite lung, the larynx and the intestine. The secondary lesion may be small,

*Read before the Section of Tuberculosis of the Massachusetts Medical Society at its Annual Meeting, June 5, 1933.

†Amberson—Assistant Professor of Clinical Medicine, College of Physicians and Surgeons, Columbia University. For record and address of author see "This Week's Issue," page 811.

or widespread, exudative or productive; it may develop and extend slowly or acutely, continuously or intermittently. Consequently the chronic advanced cases may take years to develop, but—what is much more important to realize—the fresh minimal lesion of today often is the advanced bilateral cavitary lesion of one, three or six months hence.

Before excavation and bronchiogenic spread of the early pulmonary lesion occur, there is usually a period of at least a few weeks when the golden opportunity for instituting intensive and effective treatment should be grasped promptly and with courage. Unfortunately, because symptoms and abnormal physical signs at this time are vague, indefinite or entirely lacking even though the roentgenogram shows the patch of infiltration clearly, or because the patient is reluctant and the physician timid, this day of grace is sinned away in fully seventy-five to ninety per cent of the cases.

MODES OF TREATMENT

Criteria of judgment: The ultimate measure of a system of treatment of any disease is its adequacy to restore the patient to normal living and working conditions in a minimum of time. In tuberculosis a treatment is good if it achieves local healing, abates toxemia and permits the return of normal function. But in this particular disease what is immediately good may in the long run prove to be woefully inadequate. For example, the old treatment of "superalimentation" was good because it fattened patients and put the bloom of youth in their cheeks, but, standing alone, it failed because most of the same patients were dead within five years. Indeed, no reasonably sure judgment can be passed on a treatment of tuberculosis until one has reviewed the results five years afterwards; ten is even better. There is a definite reason for this minimum five-year judgment. The patient usually has tried resuming normal living, and the durability of the local healing process has been tested. The scar of healed tuberculosis is not a healthy scar; it is a tubercle bacillus infested scar, remains so for many years, and, therefore, must stand the test of time before it can be considered sufficient. Furthermore, most of the fatalities, if they occur, are to be expected within the first five years after the termination of treatment. In an analysis of 618 fatal cases recorded by the statistical department of Trudeau Sanatorium for the years 1916-1930 and kindly furnished to me by Doctor Heise, it appears that 72.8 per cent of the deaths occurred within five years after sanatorium discharge. It is concluded, therefore, that the true results of treatment appear only at the end of this quinquennium and thereafter.

Sanatorium rest treatment: Few sanatoria keep any methodical follow-up records of discharged patients and, therefore, know comparatively little about the final efficacy of the treatment given. Trudeau Sanatorium, for one, is exceptional for the efficiency of its statistical department. In its forty-eighth annual report (1932) we see recorded in compact and valuable form the late results of sanatorium rest treatment at its best. The record of the present condition of patients discharged since 1916 shows interesting and instructive contrasts. For instance, 76.7 per cent of the patients with minimal disease on admission and 64.8 per cent of the moderately advanced are known to be well, but this applies to only 33.4 per cent of the far advanced. The contrast is even greater if we study the mortality figures of patients discharged from 1916 to 1925; followed, therefore, for a period of seven to sixteen years. Among the 107 minimal cases discharged as arrested and apparently arrested there were but three deaths or 2.8 per cent; among 183 minimals discharged quiescent, 23 deaths or 12.2 per cent; among 123 minimals discharged "active" 21 deaths or 17 per cent. The death rate amounts to 42.6 per cent in the moderately advanced "active" group of 46 patients, and finally to 67.9 per cent in the far-advanced quiescent group of 106 patients and 71.2 per cent in the far-advanced "active" group of 212 patients. (Only 13 far-advanced cases were discharged as arrested or apparently arrested during this time.) As stated, these can be taken as the results of the best sanatorium treatment of an intelligent group of patients at a time before collapse therapy was practiced to any considerable extent. It is to be expected that in less efficient institutions the results would not be nearly so good.

The seriousness of the Trudeau mortality experience is strikingly emphasized when we find that 523 or 84.6 per cent of these 618 patients died before they reached their forty-first birthday. This corresponds with the general experience that tuberculosis remains the most fatal and disabling disease in the most productive period of life. Still, many officials discourage the widening of facilities and the expenditure of public funds for the prevention and treatment of tuberculosis because, once the Captain of all the men of death, it has yielded this distinction to the maladies of old age.

So far as sanatorium rest treatment or its equivalent goes, a consideration of the Trudeau experience forces the conclusion that the results in far-advanced cases are extremely poor when measured in terms of longevity, though many patients will show temporary improvement and not a few will be enabled to live for many years as more or less chronic invalids. In the group of moderately advanced cases the re-

sults are better but still far from adequate. At the other extreme the patient with minimal disease usually responds well to rest treatment and, if he does, his prospect of keeping well under conditions of normal living are excellent.

Since comparatively few cases today come into our hands for treatment in the early most favorable stage, it is evident that we should willingly adopt and apply more widely the newer measures to improve our patients' prospects.

Miscellaneous aids: The wish for a highly potent medicine or biological agent for the treatment of tuberculosis still goes unfulfilled. Of late years there has been considerable interest abroad in the administration of gold salts, especially in the form of sanocrysin. Judging from the sentiment expressed at the last International Conference against Tuberculosis, the writer is one of a minority who do not hold this treatment in high esteem. Our experience in Detroit a few years ago, using this drug in a small group of patients, produced an unfavorable conclusion. The fact that we treated only twenty-four patients, half of whom were "controls", has been criticized. Nevertheless, we hold that the intensive study of small groups, so controlled, yields truer information about some of these therapeutic agents than does a very extensive inadequately controlled trial. It seems fair to say that the case for sanocrysin has not been proved. Agents which promote or restore normal physiological function remain, when indicated, the most useful of the general measures. These include proper balancing of the diet, heliotherapy, and the special treatment of tuberculous or non-tuberculous complications when they arise.

Bed rest: The most decided change now being witnessed in the management of tuberculous cases is the wider and more prolonged use of bed rest and the earlier and more judicious application of collapse therapy. In fact, the large number of methods and combinations of methods now available has attracted some of our best physicians and surgeons into the field of tuberculosis treatment, which is no longer a uniformly humdrum routine. This is fortunate because there is a great temptation for the inexperienced to apply some of the newer methods indiscriminately, hit or miss. The change has come from a frank recognition of the inadequacy of hygienic rest treatment in a large proportion of cases, particularly those of moderately or far-advanced disease. But in attempting to render a judgment of such treatment and to designate the point of departure to collapse therapy, we must not be misled in choosing our yardstick. In studying the follow-up figures from Trudeau Sanatorium, as we have done above, it must be remembered that these results apply to sanatorium treatment as practiced before 1925, when bed rest was not often prescribed for patients who did not have symptoms of active disease. At the Loomis Sanatorium, for instance, it was our practice around 1917 to 1920 to regulate treatment almost solely by symptomatic indications. Then, by learning how to interpret chest roentgenograms more accurately and particularly by comparing roentgenograms taken at intervals of time, our faith in symptomatic guides alone began to weaken because we found numerous disparities between symptomatic behavior and evolution of the pulmonary lesions. Patients who lost their fever, gained weight and felt better often showed new cavities or an increase in old cavities in the lungs. New spreads of the disease sometimes declared themselves in the routine serial x-ray film, days or weeks before the symptoms of renewed activity became apparent. A patient, undergoing the hardening process of graduated exercise, had a hemorrhage from a cavity which showed in the roentgenogram but gave no physical sign diagnostic of its presence. Such shocks led us to make it a methodical business always to include in our clinical routine the study and correlation of serial x-ray findings. In other words, we found a way to understand much more intelligently the behavior of tuberculosis in the individual patient and to find a congruity between symptoms, signs and actual local changes, which previously had often seemed discouragingly incongruous. This practice of bringing the findings of roentgenograms, taken every few weeks to every few months, into the clinical picture has done more in modern times to perfect the treatment of tuberculosis than any other factor. The importance of symptoms, properly correlated with the behavior of the local lesions as shown by x-ray, cannot be overstated, but the use of symptomatic indications alone for guiding treatment is to be stamped today as crude and unscientific. It is evident, therefore, that the average sanatorium treatment of 1915-1925 was not the same as that of the present. I stress this because some who urge the wholesale application of surgical measures fail to take cognizance of the much greater efficiency and scientific accuracy with which simple hygienic-rest treatment is now being practiced. For instance, the often quoted figures of Barnes and Barnes showing that 90 per cent of 1,454 tuberculous patients with pulmonary cavities treated from 1918 to 1927 were dead within five years, certainly is no fair measure of what can be accomplished with rest applied according to present concepts. Six hundred and fifty-one of Barnes' and Barnes' cases never had an x-ray. Furthermore, these workers, like many of us, were seriously hampered in those years by the lack of facilities for giving bed rest as often as they would like. While, as discussed above, cavity formation opens the way for secondary spreads of the disease and at times is the beginning of the end, it is wrong to infer that the presence of cavity practically always spells disaster. Vari-

ous authors have presented a happier side of the picture. MacMahon and Kerper in a recent study of 296 cases at the Loomis Sanatorium show that certain types of cavities if not associated with too much surrounding disease heal with great frequency on rest treatment alone. In their series, 40 per cent of the cavities not over two by two cm. in diameter closed spontaneously; but only 20 per cent of those three by three cm., and 13 per cent of those four by four cm. From a follow-up study which I have been making of cases at Loomis in which cavities healed seven to ten years ago, it appears that these patients have done extremely well under normal living conditions.

Observations like these should not delay the use of collapse therapy when it is needed. Nevertheless, in prognosticating what hygienic-rest treatment is likely to accomplish for a given case, one should remember that there are important differences in the practice of such treatment and the results will vary accordingly. Whereas, a comparatively few years ago, strict bed rest was reluctantly advised only in febrile cases, I am now convinced that the proper thing is to put promptly to bed almost every patient in whom incipient tuberculosis is found. The only exception is the patient with a lesion, newly discovered, but apparently old and fibrotic. (Even these should be checked by examination and x-ray from time to time.) In this way the degree of mildness or danger of the lesion can be ascertained under basal conditions of physiological rest and later treatment guided accordingly. Then strict rest, sometimes with weights on the chest, should be continued until healing is safely initiated and advanced—symptoms or no symptoms. When physicians learn this lesson, the lives of patients will be greatly lengthened, and our chest surgeons will more often be occupied as interested spectators.

Obviously, it is futile to hope for this happy state of affairs so long as our diagnostic machinery does not find eighty or ninety per cent of the tuberculous cases until they are advanced. Our progress on this front has been pathetically feeble, and will probably continue so until it is appreciated that the symptoms and signs of early tuberculosis are usually slight and vague and that very often abnormal physical signs are absolutely lacking, though the x-ray shows a fresh lesion. The ideal cannot be approached therefore until periodical examinations become popular and until some form of x-ray study of the lungs becomes a part of this. The unique experience of Dr. Haynes Harold Fellows at the home office of the Metropolitan Life Insurance Company where fifteen thousand employees are examined annually points a vital lesson in this respect. By including the fluoroscopic examination of the chest as a part of the procedure, Dr. Fellows and his staff have discovered the following proportion of incipient or minimal cases:

1929	28 per cent
1930	47 per cent
1931	52 per cent
1932	76 per cent

Dr. Fellows advises me that, prior to the use of his present method, 86 per cent of the patients sent to the sanatorium had symptoms and 14 per cent had none; in 1930 and 1931, 35 per cent had none; and in 1932, 40 per cent had none.

Under the aegis of Commissioner Wynne of the New York City Department of Health, a rapid x-ray case finding study, employing a paper photographic medium, has recently been made of about 20,000 people. We have discovered hundreds of cases of minimal tuberculosis. Experiences like these point the way toward the ultimate solution of a problem which has been completely baffling heretofore.

Collapse therapy: That collapse therapy is gaining in practice and is being applied more intelligently is a matter of record. If the question were asked: What is the most important action of collapse therapy in pulmonary tuberculosis? the answer would be: The closing of cavities and holding them closed until their walls fuse and heal by cicatrization. There is much to be said for functional rest and altered circulation of the collapsed lung as factors in healing, but experience is universal that the most important element in the healing process fails so long as the cavity remains open. For instance, in a series of Loomis Sanatorium cases followed by Riggins and the writer for an average of eight years after the completion of artificial pneumothorax treatment, we found 89 per cent of 82 patients, whose cavities had been closed, living, and 91.7 per cent of these were well. By contrast, of 68 cases in which the cavity was not closed, only 33.8 per cent were living and only 59 per cent of these were well. It is proper, therefore, to say that collapse therapy in the cavity case *facilitates* healing, but it it less clear just how much it *accelerates* healing. Cavities may heal more quickly principally because their walls are mechanically brought together, not so much because the fundamental healing process is greatly stimulated. It follows, therefore, and is borne out by experience that hygienic rest treatment cannot be abandoned just because the lung has been mechanically collapsed. Sometimes there is a tendency to regard tuberculosis as a purely mechanical problem, to ignore the ever-present systemic aspects of the disease, and to minimize the time element that is so essential for good durable healing, regardless of the form of therapy. Thus, we hear of the advocacy of artificial pneumothorax as soon as the diagnosis is made in order that the patient may continue with his work and home life. In a few instances this scheme will succeed just as no treatment at all will sometimes succeed, but such practice in the average case courts disaster.

A question which is gaining prominence is whether collapse therapy should be applied in the minimal or incipient case. Some advocate temporary artificial paralysis of the diaphragm by crushing the phrenic nerve or even a partial artificial pneumothorax in virtually all early cases, arguing that, if the rest "cure" will bring the desired result, additional collapse therapy will bring it more quickly and more surely. The theoretical assumption is not proof, and time and experience alone will give the true answer. My own view is that the minimal lesion without cavity formation does so well on prompt and adequate bed rest treatment and usually heals so durably that collapse therapy is not likely to make the result more sure. If one assumes that the result will come more quickly, some patients will be misled into returning to work prematurely. Few would advocate artificial pneumothorax in this stage because of the complications to be anticipated; or advise permanent paralysis of the diaphragm. Crushing of the phrenic nerve would be the procedure of choice because, with the nerve regenerating in six or eight months, the diaphragm usually regains its function. This practice is on trial in some centers and eventually we shall hope to learn the five year results in comparison with simple rest treatment.

Phrenic nerve surgery: The results of permanent diaphragmatic paralysis by phrenic nerve avulsion as performed up to ten years ago are now being reported by various authors.* The figures vary greatly, probably because of different criteria employed in selecting cases. Thus in 65 cases followed up to eight years, Mues (Berlin) found 32.3 per cent healed; 12.3 per cent improved; 15.4 per cent unimproved; and 40 per cent dead. Höring (Berlin) reports indifferent results in 56 cases and agrees with Sauerbruch and his school that adequate healing is seldom accomplished by this method alone. Harms (Mannheim) reports 100 cases operated on during eight years: 52 are dead. and of 31 survivors traced only five are clinically healed. Stekelis (Odessa) traced all but 13 of 166 cases after three years: only 14 were well; 63 improved; 28 unchanged; 14 worse and 35 dead. Patronikola (Vienna) followed 111 cases operated on from 1920-1929: eleven had other operations; 79 were dead; nine unimproved; four worse and four improved. In a group of 125 cases operated on six months to four years previously, Oekonomopoulo reports 40 healed and 66 able to work. Generally speaking, these figures are disappointing. Our experience is that little or nothing can be expected from phrenic nerve interruption or avulsion alone in extensive tuberculous lesions of any type. It does not seem to influence favorably the very active exudative or caseous types of disease, nor do cavities close following the procedure if they are thick walled or widely attached to the parietal pleura. As mentioned by Douglas and others in recent articles the rather fresh, thin walled, not adherent, middle or lower field cavity, without too much infiltration around it is most likely to be favorably affected. This procedure has a definite place but must be applied with much more limitation than it was five or ten years ago. Because of the uncertainty of the effect and the disadvantage of a permanently paralyzed diaphragm in relation to respiratory and circulatory function in later years of life, there is a properly growing tendency not to avulse the nerve but only to crush it, repeating or amplifying the operation after six months if indicated.

Artificial pneumothorax: This is still the most widely applied form of collapse therapy, because, for the general run of suitable cases, it is the surest means available to set the stage for durable healing. Nevertheless, it is not used nearly so often as it should be. Physicians too often satisfy themselves with bringing about mere abatement of symptoms and quiescence of the disease. They fail to take the long view and to give due weight to the fact that, even with symptoms receding and constitutional condition improving, inadequate healing of the pulmonary lesion is a snare which eventually proves disastrous. Furthermore, the patient is even less able to sense the lurking danger in this state of improvement. More often than not he will terminate his treatment prematurely, unless a knowing physician in whom he has confidence can convince him of the truth. As long as physicians concern themselves merely with securing immediate symptomatic amelioration without giving due weight to the pulmonary status as demonstrated principally by the x-ray and the presence or absence of tubercle bacilli in the sputum, as long as the plan of treatment looks ahead only for a year or two rather than for thirty or forty years, just so long will we find a large proportion of our moderately advanced and many of our early cases disabled or dead in five to ten years. Only when our present knowledge is turned into practice will these people enjoy the span of life to which, in human judgment, they should be entitled.

In looking at pneumothorax we must of course weigh its complications and disadvantages many of which are now being avoided or controlled by proper technique and management. Its practice should not be left in the hands of novices. The technique is simple, but the general management of the case merits the thought of the experienced clinician, who has the prognostic ability to picture with reasonable probability the ultimate fate of each patient. Since we have discarded the useless or harmful ingredients of old formulas of tuberculosis treatment, we give pneumothorax not only to the obviously

*For the purpose of this paper I have gone over the accounts of the various forms of collapse therapy as reported in the German abstract journal, *Zentralblatt für die gesamte Tuberkuloseforschung*, for the past two years The articles cited are from this source.

progressive unilateral pulmonary case, but also to the cavitary case which is not healing even though the disease is not progressive. The interval of preliminary rest treatment is now omitted or shortened in the case in which past experience has shown a poor chance of ultimate recovery. This practice is reducing the period of invalidism, which is ruinous to the patient and costly to his family or community, not to speak of lengthening life.

The wider use of pneumothorax has necessitated a proportionate development of accessory measures. For instance, in overcoming the troublesome interference of adhesions the Jacobaeus operation for direct severance and the use of pressure pneumothorax for stretching can be practiced with much advantage. At present we favor the latter procedure for most cases, since it is usually safe and often effective if properly managed.

Bilateral simultaneous artificial pneumothorax, while it sometimes brings remarkable results, is difficult to manage and less promising with regard to late recovery and survival.

Oleothorax: Investigations which we have been conducting at Bellevue convince us that the early claims for the benefits of instilling oil, medicated or not, into the pneumothorax space were too sanguine. Nevertheless, the treatment has some merit in controlling the troublesome tuberculous effusions and empyemas which so often complicate pneumothorax treatment. Our experience will be reported later.

Thoracoplasty: For the uncomplicated unilateral fibroid cavitary case, in good general condition and with good cardiocirculatory function, the results of thoracoplasty by rib resection are remarkably fine. This is the type of case which Archibald calls the "good chronic". Pneumothorax usually fails of its purpose because of widespread pleural adhesions. With the perfection of case selection, operative technique and supplementary operations, the original high postoperative mortality has been reduced to about a ten per cent average. Supplementary operations are mentioned because lateral costectomy or some other revision may be necessary to accomplish the closing of cavities and one should consider this possibility when advising radical surgery. It is, of course, unfortunate that a good many patients are obliged to face such drastic treatment because the earlier opportunities for easier and more promising therapy were neglected or poorly applied. But, thanks to surgeons, the outlook has improved vastly for these advanced cases. Jessen (Davos), for instance, has had no postoperative deaths in the cases with ideal indications, and reports healing in 81 per cent. But, as he points out, just as soon as one deviates from the ideal the mortality rate goes up, and in the manifestly unfavorable group of cases mounts to 50 per cent within two months after operation. In Detroit, Dr. Chadwick informs me that O'Brien has performed 63 thoracoplasties since August, 1932, with a postoperative mortality of only 1.5 per cent. The figures of Jessen and O'Brien indicate that the best results are obtained in special hospital departments or sanatoria where the experienced physician has an important share in the selection and management of the cases. In a crowded hospital like Bellevue where facilities and personnel are greatly overtaxed, our experience has at times been most discouraging. As to ultimate results, the experience of ten years with 80 cases at Loomis Sanatorium corresponds closely with that which Nissen reports for Sauerbruch's clinic and others, viz., 30 to 40 per cent can be expected to get good durable healing and 30 to 70 per cent (including the latter) some improvement. Forty per cent of the Loomis thoracoplasty cases are dead. Time, no doubt, will improve these prospects still further.

Other operations: Severing the scalenus muscles, paralyzing the intercostal nerves and ligating the pulmonary vein all may aid in healing a tuberculous lesion but, with very few exceptions, these measures seem to offer little promise because they fall short of closing cavities, which is the main problem. Recently, Durante (Genova) has advocated cutting the long thoracic nerve to paralyze the serratus muscle in connection with cutting the scalene muscles and phrenic nerve—a most doubtful practice.

Partial thoracoplasty for apical cavitary tuberculosis is often effective when pneumothorax fails or is disadvantageous. This, with or without an extrapleural freeing and compression of the pulmonary apex by a temporary medium such as rubber dam (Lilienthal) seems preferable to the paraffin plombe. Experiences, such as those of Denk (Vienna) and Arnold Winternitz, show that complications from the permanent sojourn of this foreign body in the chest —particularly rupture into a bronchus—are not inconsiderable.

CONCLUSION

The changing aspect of tuberculosis treatment is a hopeful thing. Rest is still the mainstay, but the old practice of prescribing it solely according to symptomatic indications was faulty and often disastrous.

Strict bed rest is often needed for weeks or months after all constitutional symptoms have subsided. Its regulation then depends greatly on a knowledge of the intrinsic state of the local lesion as revealed best by serial x-ray examinations. The faithful application of this truth, especially in the early case, will go far to avoid the necessity of radical surgery.

Nevertheless, collapse therapy should be practiced much more widely and more promptly when rest treatment alone offers little prospect of ultimate complete recovery. Physicians can

now prognosticate the probable outcome more accurately than ever before.

The need is for medical and surgical judgment to assure the most discriminating and judicious application of the many forms and combinations of treatment now available.

Discussion

Chairman Morgan: The discussion of this paper will be opened by Dr. Emerson.

Dr. Ernest B. Emerson, Rutland, Mass.: *Mr. President and Members of the Tuberculosis Section*—It is difficult to add much to what Doctor Amberson has said in his most excellent paper. We have made tremendous progress in the treatment of tuberculosis during the past few years, yet in some ways we have fallen down, particularly in diagnosis which is so essential for treatment.

A few weeks ago a patient gave me these figures; take them for what they are worth: of 238 cases interviewed in the sanatorium, approximately 30 per cent had been wrongly diagnosed. The average delay in treatment from the time these patients went to their first doctor to the time they arrived at Rutland was forty-one weeks. They visited from one to five different physicians. These are the diagnoses: bronchitis, rundown, colds, pleurisy, pneumonia, stomach trouble, liver trouble, low blood pressure, asthma, tonsillitis, nervousness, and imagination.

The National Tuberculosis Association in 1914 made a survey of sanatorium admissions. In 1914, ten per cent were reported as minimal; ten years later, 14 per cent; and in 1928, 12 per cent were minimal cases. About 11 per cent of the admissions to Rutland last year were classified as minimal cases. Apparently there is little improvement in diagnostic methods.

All of us doing tuberculosis work know that it is frequently a difficult matter to make an early diagnosis. It is for this reason that the State and County Sanatoria have established diagnostic clinics to which patients may be referred by the family physician and a report sent to him directly. It would seem that if greater use were made of the diagnostic clinics, that more early cases would be sent to the sanatoria. A step further would be a change in the law permitting the admission of patients to sanatoria for observation as in the general hospital.

Not so long ago the treatment of tuberculosis was exercise, fresh air, and stuffing, and these ideas still persist. Little was said about rest. Today rest is considered the corner-stone of whatever may be done. For the average individual this can be carried out better in the sanatorium than at home, and what is more valuable than anything else, broadly speaking, is the education of the patient. I believe the educated patient who comes out of a sanatorium is an apostle of good health and does a great deal of good, and certainly he cannot or will not take or continue intelligent treatment unless he has been instructed.

Collapse therapy has restored to a reasonable degree of health many advanced cases. It is equally applicable to many of the early cases. It has been brought out that collapse therapy is only supplementary to rest treatment. I think this point should be emphasized. There seems to be some feeling that if a case can be operated upon that that is all there is to it; whereas, the fundamental principles of treatment—rest, proper food, and clean air, are absolutely essential for the success of whatever surgery is done. Thoracoplasty or other radical surgical interference should not be attempted except after careful observation on the part of both internist and surgeon. Neither one alone has all the qualification for selecting the case. The importance of team-work should not be overlooked. The results of surgery, good as they are, should not obscure the importance of rest treatment. With regard to the closing of cavities, I can see no reason why in the majority of instances we should waste time attempting to stretch an adhesion to permit closure of a cavity when these cavities will close promptly following a pneumolysis. It is true that pleurisy may follow the operation; however, in our experience this has not been a disastrous complication. Our experience with phrenicectomy has been rather disappointing. We have not seen the results reported in other clinics. Artificial pneumothorax is our first choice. We are collapsing earlier and in more cases as time goes on. A few years ago it was necessary "to sell" artificial pneumothorax to the patient. Today, convinced of the results, he is demanding it or some other form of surgical treatment. We have been accused of being a little radical in the matter of collapse therapy, inasmuch as at the present time about 61 per cent of the patients are receiving it in some form.

Doctor Chadwick informed me that 75 per cent of the patients in his clinic were under some form of collapse therapy and stated that it is his belief that probably 80 per cent of sanatorium patients were suitable for this treatment. Whether or not this is so, I can see no objection to attempting pneumothorax in many cases hitherto considered unsuitable. Occasionally one of them is benefited. If no improvement is observed or the symptoms are aggravated, discontinuance of the treatment has not been followed by any further aggravation of the original condition.

With regard to the point brought up by Doctor Mahoney, I believe it is a mistake to undertake the treatment of these cases by a rotating

staff, particularly when the rotation comes every month or two. Little more than a diagnosis can be made when a change of service occurs and no one can develop a real interest in the problem or be held responsible under such conditions.

CHAIRMAN MORGAN: The paper will be open to general discussion.

DR. GERARDO M. BALBONI, Boston: Naturally there are some points that Dr. Amberson did not mention in the treatment of pulmonary tuberculosis by artificial pneumothorax. Some have said that pneumothorax treatment is easy to do. I consider the carrying out of the treatment a long tedious procedure. To follow a patient along means hard work and great enthusiasm on the part of the doctor and great persistency on the part of the patient, for there are many pitfalls and the treatment is long.

The treatment of severe one-sided pulmonary disease with collapse of the lung by artificial pneumothorax belongs to the methods of treatment that we have gotten directly from nature. James Carson, a physiologist, in 1819 advocated collapse therapy, and under his supervision and in two cases of advanced pulmonary tuberculosis, artificial pneumothorax was attempted by the incision method. Both cases died, no pneumothorax was created and at autopsy both cases showed an adherent pleura.

It was not until the time of Forlanini in 1882 and on, that the present method of collapse therapy was properly established, especially on account of the failure to discover a specific for the disease. German clinicians and others followed in the footsteps of Forlanini. Collapse therapy has become definitely more valuable with the x-ray and many have contributed toward making the method safer and more valuable.

In a practically unilateral case with or without cavitation where the lung can be properly collapsed and maintained so, and the contralateral lung develops no activity, that patient will get well. We have cases that are well that were treated twenty-two years ago at the Massachusetts General Hospital and in private practice.

A partial collapse will not cure pulmonary tuberculosis. You must have a proper collapse and maintain it at a certain volume and tension, not at a positive tension, necessarily, but at the optimum tension suitable to that case. Each case is an individual case and has to be followed individually, not as a rule, as a group. If you collapse the lung properly and maintain it collapsed, not for one month, not for two months, but for years, you will obtain results. Our cases have been kept collapsed from one year to six years. The lung gradually reëxpands, never fully though, anywhere from fifty to eighty per cent. The patients are perfectly well, attending to their regular duties.

In a partially collapsed lung, where pathological sounds and breathing persist, the disease will continue and spread. It was demonstrated by Forlanini first, by others later and in two of our cases at the Massachusetts General Hospital coming to autopsy after the diseased lung had been kept collapsed for two years, no new disease had developed in the collapsed lung and the old diseased parts had healed by fibrosis.

Where a diseased lung cannot be collapsed properly on account of adhesions, it is wrong to attempt to loosen these by increased pressure. Harm will be done, perhaps tear lung parenchyma, a bronchial fistula develop or pyopneumothorax.

Adhesions that can be cauterized by the Jacobaeus method will bring about the proper collapse. We have cases where that has been done by Dr. Churchill and the patients are doing well.

We have the case of a young woman with extensive disease where one side was first treated, the adhesions cauterized and then a proper collapse was obtained; then increased activity developed on the other side, this was treated, here adhesions prevented a proper collapse and these were cauterized with excellent results. This patient is doing well, both lungs are kept collapsed. She has now very little sputum and the last three sputum examinations showed no tubercle bacilli. She comes to the hospital every three weeks for fluoroscopic examination, and receives treatment at home every week. Of course, not many bilateral cases will do that, nevertheless this case is encouraging for us to keep on with and attempt others. This particular case had been kept in bed nine months and was getting worse. We are hopeful of getting her entirely well.

As to the time of beginning artificial pneumothorax, I am glad Dr. Emerson spoke of that. It should be done earlier than it has been done. It has proved its value in certain far-advanced cases. Why keep a patient in bed months and perhaps lose the chance to give beneficial artificial pneumothorax?

It is wrong to tell a patient that pneumothorax treatment is absolutely necessary and that without it, he cannot recover; one never knows if the attempt will be successful. The attempt is the thing that decides. Except in definite indications, such as hemorrhage, etc., one should not attempt to undertake pneumothorax without sufficient observation of the patient beforehand.

Artificial pneumothorax does not exclude other forms of treatment. It may be an adjunct to phrenicectomy, lobectomy, rib resection, etc. Pneumothorax will not benefit all cases but will benefit and cure many.

Dr. Samuel Robinson, from whom I learned the technique, has been really our great exponent here. He brought it from abroad in 1909 and with Cleaveland Floyd did the first cases in

the tent ward at the Massachusetts General Hospital and we have carried it on ever since.

DR. MAHONEY: In regard to the discussion of Dr. Emerson, he said that he was very sorry that cases that go to Rutland are received so late. I wonder if anybody has had the same experience that we have had in trying to get cases into Rutland. They are usually put on a waiting list and that "waiting" extends over months and years and perhaps after the death of the patient.

Now relative to the house officer at our tuberculosis hospital, he has been on duty there for ten years without a substitute. In my opinion, he certainly is capable under proper stimulation which I think should come from the State Department of Health, relative to the prognosis of adult tuberculosis, of doing better work. It is not a month to month service.

CHAIRMAN MORGAN: Any further discussion?

DR. EDWARD D. CHURCHILL: I think that some one should speak from the surgical point of view. The paper of Dr. Amberson was a very lucid exposition of the subject grounded on sound clinical experience. I am in total accord with most of his statements. The extent to which surgical methods in the treatment of tuberculosis have been applied varies tremendously from place to place. We all know that there are certain centers in this country and certain centers abroad that are exceedingly active in extending application of surgical methods and artificial pneumothorax. There are also certain communities that have been very slow to accept these forms of treatment. I personally feel, being familiar with the work in other parts of the country, that our community—I refer to Massachusetts as a whole—has not as yet reached the optimum use of collapse therapy. We need harbor no fears at the present time that the pendulum has swung too far. I know that there are still a great many cases close at hand that could be restored to active, wage-earning capacity, who are being kept as institutional patients at a terrific cost to their local boards of health. When progress in the treatment of any disease reaches a certain point, those who make use of the newer forms of therapy really become the conservatives; whereas doctors who continue to pin their faith to an older method, even though it be a policy of watchful waiting, deserve the criticism of being unduly radical.

SPEAKER FROM THE FLOOR: *Mr. Chairman*—My experience is rather limited, but, in the cases that I see, certain classes are certainly going radical. My point is that a man makes a diagnosis of tuberculosis and reports it.

But some local surgeon comes in and does phrenicotomy. I have had cases with positive x-ray findings and the results have not been good but they are few.

I also want to back Dr. Emerson in that diagnosis respecting bronchitis. I think that one who is doing chest work should emphasize that idea. Bronchitis is not really a disease; it is more of a symptom. There is some underlying infection underneath. It is like jaundice. That diagnosis of chronic bronchitis continuing, I think leads us to many of our advanced cases.

CHAIRMAN MORGAN: Is there anything more to be said? If not, will you close the discussion, Dr. Amberson?

DR. AMBERSON: *Mr. Chairman*—All I would like to say in closing is to thank you gentlemen for the discussion and say that our interest in collapse therapy is remotely, at least, due to the interest which was created here in Boston in the early years. Dr. Cleaveland Floyd came down to Loomis in 1912 and showed us how to perform pneumothorax.

THE PATHOGENESIS OF MULTIPLE SCLEROSIS: A POSSIBLE VASCULAR FACTOR*

BY TRACY J. PUTNAM, M.D.†

IT is now but a few years less than a century since neuropathologists began to puzzle over the nature of that strange disease multiple sclerosis. The great French pathologist Cruveilhier described and illustrated the curious irregular scars "almost like areas of embolism", which occurred in the central nervous systems of two patients both of whom had suffered from a slow progressive paraplegia and tremor before death[1]. Multiple sclerosis is now recognized as one of the commonest diseases of the central nervous system, and its pathology and clinical course have become very familiar to all neurologists, but there is still no unanimity of opinion as to whether it is infectious, neoplastic, toxic, hereditary or degenerative in origin.

I have no really new observations to report on the clinical aspects of multiple sclerosis. We all realize that Charcot's triad of nystagmus, scanning speech and intention tremor composes the picture of a late stage of the disease, and that many patients present themselves with no more than mild paresthesias of one extremity, temporary diplopia, or blurring of vision, who later display unmistakable manifestations of multiple sclerosis. I should merely like to call your attention to certain symptoms which have appeared rather frequently in my experience, but which are not often mentioned in the ordinary textbook discussions of the subject.

The first of these is pain[2], which may be postural, usually backache, from muscular weakness, or may be radicular, of which the most striking example is the true trigeminal neuralgia occasionally seen with multiple sclerosis. The second somewhat unusual symptom is convulsions which I have seen in three instances. The third is papilledema which may lead to an erroneous diagnosis of brain tumor. The fourth is a Parkinsonian syndrome. In brief, there is scarcely any neurological disease which may not be simulated by multiple sclerosis. The diagnosis must rest, of course, on finding evidences of lesions scattered through time and space in the central nervous system and a history of exacerbations and remissions.

I should like to turn at once then to the pathology and pathogenesis of the disease, which have constituted the field of my own investigations under the Harvard Multiple Sclerosis Fund. The histology of the lesions is now well established. Indeed, the essential histological changes were described by Charcot in 1868[3] as consisting of a patchy loss of myelin, with relatively intact axis cylinders, perivascular infiltration, and dense glial proliferation. These observations were made on teased specimens of tissue, for the microtome was not in use, and with no better stain than carmine. Our elaborate modern histological technique has added nothing essential to this description, but the examination of an increasing number of cases has shown that a whole series of different histological pictures may be observed in which the myelin is predominantly involved and the axis cylinders relatively intact, ranging from the obviously chronic state of multiple sclerosis with a dense glial scar and little or no perivascular infiltration to the obviously acute encephalomyelitides, of which the postvaccinal and postmeasles encephalitis are the most familiar forms, with massive lymphocytic perivascular infiltration and little or no actual sclerosis. Some such cases have been described as "acute multiple sclerosis", and sometimes acute and sclerotic lesions have been observed in the same case, but eminent authorities have expressed doubts as to whether it is justifiable to assume that the acute stage would have developed into the chronic one if the patient had survived. Histological examinations have been reported in three patients dying months after postinfectious encephalitides. In one of these, multiple cysts of softening with complete loss of axis cylinders was found[4], entirely unlike multiple sclerosis; in the other two, sclerotic lesions with relatively intact axons[5].

When I first approached the subject of multiple sclerosis, it seemed to me that the two fundamental questions were the following: does the process of demyelination such as is seen in the acute encephalomyelitides regularly or ever lead to chronic sclerosis? and if so, what is the nature of the acute process? The former question seemed to me to be the one to undertake first, and I felt that if it could be approached experimentally, a more conclusive answer might be obtained than that deduced from chance observations at autopsy.

A survey of the literature revealed that experimental lesions characterized by areas of myelin loss in which axis cylinders remain practically intact have been produced in two apparently quite dissimilar ways: by exposure to carbon monoxide[6], and by injection of small amounts of tetanus toxin[7]. Both these methods are extremely uncertain. Of sixteen animals which we treated with carbon monoxide to the point of coma, only two showed satisfactory le-

*Address delivered before the Hartford Medical Society, February 20, 1933.
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sions (figs. 1 and 2) and only two out of eighty dogs injected with tetanus toxin developed symptoms referable to the central nervous system. The details of these experiments have been recorded elsewhere, and for the present I wish only to call attention to the fact that in both instances the myelin was destroyed and taken up by phagocytes, the axis cylinders remained almost intact, and there was a perivascular infiltration of fat-cells, lymphocytes, and plasma cells. In lesions which had lasted for some months, there was also gliosis. Naturally, the gliosis was not so intense as that seen in lesions from patients who have had their disease many years, but all of the features of multiple sclerosis upon which the pathological diagnosis is based were present.

FIG. 1. Myelin lesion in the white matter of a dog's hemisphere simulating that of multiple sclerosis in experimental carbon monoxide poisoning. Weigert-Pal stain[18].

Experimental evidence is therefore added to the somewhat ambiguous pathological evidence that acute lesions in which myelin is damaged without disturbance of the fundamental architecture of the tissue, may evolve into the glial scars which are characteristic of the fully developed stage of multiple sclerosis. But what common denominator can exist between carbon monoxide poisoning, tetanus intoxication, and the insidious progressive disease with fairly characteristic course which we recognize in human beings?

I need not pause over the multitude of conceivable possibilities which my co-workers and I investigated and discarded, before recognizing that in all instances the lesions had a predominantly perivascular distribution, as has long since been recognized in multiple sclerosis[8] and that in some of them, actual thrombi could be demonstrated. The histological picture is of course entirely different from that ordinarily seen, following thrombosis or embolism of a cerebral artery, which usually leads to destruction of axis cylinders, glia and all nervous elements within the anemic area, followed by the formation of a cyst with connective tissue walls or a mesodermal scar. In the plaques under discussion, the lesion is a much milder one, so mild that the axis cylinders and connective tissue, more resistant than the myelin, are but little affected, and there is little stimulus to mesodermal proliferation.

The clue to a possible solution was found in the demonstration by Pfeifer[9] that around many small vessels in the brain there occurs a zone almost devoid of capillaries, which may be three to four times the diameter of the vessel itself. The inference is that the tissue in this area may be chiefly nourished by transudation through the walls of the vessel, and it seemed conceivable that obstruction of a *length* of vessel might cause a relative anemia of the surrounding tissues, without being sufficient to cause an area of necrosis because of a supply from neighboring branches.

In the hope of producing such a condition experimentally, a commercial emulsion of cod-liver oil was employed, in which the globules were of various diameters up to about 0.5 milli-

meters, which is more or less the size of the vessels around which "capillary-free" spaces occur. This mixture was injected into the carotids of cats which were allowed to live varying lengths of time up to several months.

The results were both encouraging and disappointing. Tiny lesions were sometimes produced with all the characteristics of sclerotic plaques. More often, however, small or large cysts of softening occurred, with all transitions between the two types. In general, the lesions were much more like those seen with the acute post-infectious encephalitides which come to autopsy within a few weeks of the onset. This confirmed our impression that the histological features of plaques might be produced by interference with blood-supply, but on the other hand, made it seem improbable that the lesions of the human disease were produced by embolism or arterial thrombosis.

Here the matter stood until a quite different set of experiments suggested the next step. With Dr. Eric Ask-Upmark, I have been investigating the choroid plexus of the living cat microscopically. In the course of our observations, we watched the vessels of the plexus and adjacent white matter under an ultraviolet light during the intravenous injection of fluorescein. Fluorescein is an extremely diffusible dye which may be detected in very small amounts by its yellow fluorescence in light of short wave length. It has already been used to trace the source of the spinal fluid[10] but in these experiments, we had an exceptional opportunity to observe exactly where its diffusion was greatest. This was not through the dense capillary plexus, but through the smaller veins of the choroid and white matter. Physiological investigation therefore confirms Pfeifer's conclusion from anatomical observations that the "perivascular cap-

FIG. 2. The same lesion stained from an adjacent block by the Bielschowsky method for axis cylinders, which are practically intact[18].

illary-free space", and perhaps a still larger zone, obtains part of its nourishment from the vessel contained within it. Pfeifer had also pointed out that this vessel was usually or always a vein. On reviewing the slides of our experimental animals and also some from cases of human multiple sclerosis, I came to the conclusion that when a lesion was definitely perivascular in location the central vessel could usually be identified as a vein, never as an artery, according to the criteria laid down by Pfeifer. The tentative hypothesis seemed to be justified that the lesions were produced by a venous obstruction.

For some time, no method of testing this hypothesis experimentally presented itself. Very recently, however, I have been able to produce some acute lesions which have both gross and microscopic resemblance to those occurring in human beings by injecting bland oily substances forwards—that is, upstream.—into the ligated longitudinal sinus of dogs. The resulting lesions are confined almost exclusively to the white matter, and consist of gradual myelin changes, a perivascular infiltration with phagocytes and lymphocytes, and a glial pro-

liferation. There is no destruction of axis cylinders or of connective tissue structures, and only minor changes in ganglion cells (fig. 3). The changes therefore resemble the milder fresh plaques of human multiple sclerosis. It remains, of course, to be seen whether the late stages will also show a characteristic structure.

FIG. 3. Myelin lesion in the white matter of a dog's hemisphere simulating multiple sclerosis produced by experimental venous obstruction. In the same lesion, axis cylinders are intact and there is a glial proliferation. Weigert-Pal stain.

As a matter of fact, the idea that the plaques of multiple sclerosis are dependent on venous thrombosis is not new. It was suggested years ago by Ribbert[11], though without conclusive proof, and many pathologists have described thrombi and intravascular changes within the sclerotic areas. That other pathologists have failed to find them is not surprising, even if this hypothesis is correct, for we should expect them to become organized or recanalized in old lesions, so that even traces of them might be hard to find. In some specimens under study at present, it appears that a roughly normal number of capillaries are present in the plaques as compared with corresponding normal areas, but that only a small proportion of them are patent.

It may be objected that if sclerotic plaques are due to venous obstruction, such a condition should be demonstrable as a result of known vascular lesions. Actually a similar condition has long been recognized. Binswanger and Alzheimer[12] first described a disease which they called "encephalitis subcorticalis chronica", in which large areas of demyelination with persistence of axis cylinders develop on an obviously arteriosclerotic basis. Both they and investigators using the most modern histological techniques[13] agree that the condition closely resembles multiple sclerosis pathologically.

It is premature to attempt to correlate the pathological processes here outlined with the recognized features of the human disease in any detail. Infections are known to predispose to the production of thromboses in various parts of the body, and also to the onset or exacerbation of multiple sclerosis. The association, both pathological and clinical, between multiple sclerosis and retrobulbar neuritis, and between the latter and sinus disease[14], is well recognized. Exacerbations of multiple sclerosis are almost constantly produced by pregnancy, in which the coagulability of the blood is increased. Obviously, new methods of clinical studies are urgently needed to demonstrate a possibly increased tendency to thrombosis.

If this hypothesis of the histogenesis of the lesions of multiple sclerosis can be maintained and, so far no definite contradictory evidence has appeared, most of the current conjectures concerning the etiology of the disease will have to be abandoned. The supposition that there is an infectious agent, a microbe or virus, actually present in the lesions becomes superfluous if similar plaques and "inflammatory" infiltrations can be produced by mechanical interfer-

ence with the blood supply. Nor need a specific "myelinolytic" toxin or inorganic poison be postulated. As a matter of fact, the evidence in favor of the importance of all of these agents is incomplete. One investigator is almost alone in demonstrating occasional spirochetes[15]. The pleomorphic streptococcus which can cause multiple sclerosis as well as poliomyelitis, torticollis and hiccups can no longer be taken seriously[16]. The theory that multiple sclerosis was caused by a filtrable virus, *spherular insularis,* has been retracted[17], and finally, an immense number of inoculation experiments, including many on apes under favorable conditions, have remained negative.

In regard to treatment, no positive advances have been made. It is but natural that a rational therapy is impossible before etiology is understood. Most of the empirical methods of treatment, fever therapy, arsenicals, antimony compounds, high vitamin diet, x-ray treatment, ultraviolet light, forced drainage of the spinal fluid, and quinine have been thoroughly tried at this clinic and others in this vicinity without more success than can be accounted for by the hope which they engender in the patient's mind. Our best results have been obtained by a candid psychotherapy and by graduated exercises, by a policy of determining to do the best possible with what is left, rather than by any artificial suggestion. In female patients, pregnancy should certainly be avoided.

SUMMARY

1. Multiple sclerosis is a protean disease, which may simulate almost any lesion of the central nervous system.

2. The histological characteristics of the disease may be closely imitated experimentally by a number of different procedures, all of which have in common an interference with the blood supply of the affected area. These facts give a new importance to the vascular abnormalities which have long been recognized as a characteristic of sclerotic plaques.

3. If the specific lesion is produced by a simple mechanical interference with blood supply, it is superfluous to postulate a specific demyelinizing organism, virus, or toxin to account for such lesions. The ultimate etiological factor should probably be sought in a local vascular abnormality, or in some alteration in coagulability of the blood.

NOTE: Since the above was written, Ferraro[19] has demonstrated that myelin lesions resembling those of diffuse sclerosis may be produced by injection of cyanides. This lends further support to the idea that various forms of asphyxia may damage myelin more than they do other tissue elements.

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HONORS TO THE DISCOVERER OF DIPHTHERIA ANTITOXIN

A celebration of the twentieth anniversary of the first production of toxin-antitoxin for the prevention of diphtheria will be conducted on October 24, in New York by the medical societies in the Bronx and Queens, and health officials of the two counties.

Dr. William H. Park, director of the bureau of laboratories of the Department of Health, a pioneer in the work of diphtheria prevention in this country, will be honored at both gatherings as will Dr. Béla Schick, discoverer of the test by which it is possible to determine the immunity of an individual to diphtheria.

Memorials signed by thousands of immunized children in the two boroughs will be presented by Dr. Park.

According to the Department of Health, more than 1,000,000 children have received immunization treatment in the last four years, and it is estimated that about 500,000 are yet to be immunized. This work will go on until the rest of the 80,000 unimmunized children in Bronx and Queens counties have been protected.

NEW ENGLAND SURGICAL SOCIETY

ANNUAL MEETING

THE New England Surgical Society held its sixteenth Annual Meeting in Boston, September 29 and 30, 1933. On Friday morning operative and dry clinics were held at the Massachusetts General Hospital, the Boston City Hospital, the New England Deaconess Hospital, the Peter Bent Brigham Hospital, the Baptist and the Beth Israel Hospitals by their respective Staffs. At noon the members met at lunch at Vanderbilt Hall. In the afternoon the following Scientific papers were presented in the amphitheater of the Harvard Medical School:

Carcinoma of the Small Intestine
Dr. Horace K. Sowles

Operative Management of Cancer of the rectum
Dr. Frank H. Lahey
Dr. R. B. Cattell—by invitation

Cancer of the Breast, End Results: Massachusetts General Hospital, 1921, 1922, 1923
Dr. Robert B. Greenough; Dr. Grantley W. Taylor, and Dr. Charles Dumas—by invitation

Cancer of the Breast, End Results; Massachusetts General Hospital, 1924, 1925, 1926
Dr. Channing C. Simmons; Dr. Grantley W. Taylor, and Dr. Richard W. Wallace—by invitation

Regional Ileitis; a Clinical, not a Pathological Entity
Dr. John Homans
Dr. George M. Hass—by invitation

Abdominal Symptoms With or Without Abdominal Lesions in Diabetic Acidosis
Dr. Leland S. McKittrick

Spontaneous Rupture of the Stomach During Labor
Dr. James Raglan Miller

Some Further Observations upon Contamination of Operative Wounds by Air-Borne Bacteria
Dr. Ernest L. Hunt

In the evening the Annual Dinner was held at the Harvard Medical School, at which Dr. Lyman Allen of Burlington, Vermont delivered his Presidential Address, using as his topic The Early Medical History of Vermont. There were no other formal speeches. Ninety-two members were present.

On Saturday morning the Scientific program was continued at the Medical School as follows:

The Diagnosis and Treatment of Acute Sub-Dural Haematomata. A Report of 34 Cases.
Dr. Donald Munro

Notes on Giant Cell Tumors of Bone
Dr. Frederic Jay Cotton

A Study of Two Hundred and Thirty-six Compound Fractures Treated at the Massachusetts General Hospital.
Dr. Ernest M. Daland

Rupture of the Intervertebral Disc
Dr. William Jason Mixter
Dr. Joseph Barr—by invitation

Compression Fractures of the Vertebral Bodies
Dr. John P. Bowler
Dr. John F. Gile

Appendices Epiploicae
Dr. Daniel C. Patterson

Suppurative Thyroiditis
Dr. Robert C. Cochrane

At the Annual Meeting which followed, the following officers were elected:

President: Frederick B. Sweet, M.D., Springfield, Mass.
Vice-President: Channing Simmons, M.D., Boston.
Secretary: John M. Birnie, M.D., Springfield, Mass.
Treasurer: Peer P. Johnson, M.D., Beverly, Mass.
Recorder: Walter G. Phippen, M.D., Salem, Mass.

The report of the Fracture Committee was read by Dr. Charles L. Scudder.

It was announced that the following members had died during the year:

Eugene B. Eastman, M.D., Portsmouth, N. H.
William P. Graves, M.D., Boston, Mass.
Sir Robert Jones, M.D., Liverpool, England.
George H. Monks, M.D., Boston, Mass.

The wives of the Boston members entertained the visiting wives at tea at the Chilton Club on Friday afternoon.

ANNUAL REPORT OF THE FRACTURE COMMITTEE OF THE NEW ENGLAND SURGICAL SOCIETY

BOSTON, MASSACHUSETTS, SEPTEMBER 29, 1933

YOUR Committee has been active during the past year in furthering that for which the Committee was originally formed, namely, the taking of direct instruction in the treatment of fractures to the doctors of New England.

Your Committee presented the move contemplated, to the regular meeting of the New England Medical Council.

The New England Medical Council is composed of the following from each of the six New England States: The President, The Secretary, and three members of the State Societies. Your Committee felt that the coöperation of this representative group would help in the carrying out of its program. The council unanimously approved the plan proposed.

The Commonwealth Fund has been approached by your Committee with the offer to coöperate with the Fund's endeavor to carry medical instruction to the country doctor. The Fund thinks it is necessary that each State should make a contribution of money to the movement, to substantiate its approval of the scheme. Therefore, the several State Societies were asked to appropriate money for this purpose.

The Massachusetts Medical Society voted part of	$500.00
The Vermont State Medical Society voted	500.00
The New Hampshire Medical Society voted	500.00
The Rhode Island Medical Society voted	50.00
The Maine State Medical Association voted	0
The Connecticut State Medical Society voted	0

Through a failure to make the plan of the Committee clear, the coöperation of the states of Maine and Connecticut to the extent of contributing was not obtained.

We are quite sure that both these states will assist and coöperate completely in this move when they understand that it will in no way interfere with State activities.

These contributions have been made known to the Commonwealth Fund. A formal application to this Fund has been made for help to carry out the plan of your Committee in New England. We are awaiting the report of the Commonwealth Fund.

The amount asked for from the Fund is $10,000.00.

Your Committee asks that it be continued.

Respectfully submitted,

ARTHUR W. ALLEN, *Secretary.*

Committee:
Charles L. Scudder, Chairman,
Robert J. Graves,
Arthur W. Allen, Secretary.

A SKETCH OF VERMONT'S EARLY MEDICAL HISTORY*

BY LYMAN ALLEN, M.D.†

IN casting about for a subject upon which to address you I remembered the pleasure with which I had listened to some historical papers upon the early physicians of Rhode Island and Massachusetts, read by former Presidents of this Society, and I thought that a similar paper telling of the early physicians of my own state would be interesting. However, I soon realized that Vermont had no really early history. The state was scarcely settled at all until 1763 and a mere 170 years is as nothing to you who proudly trace your beginnings to the early 1600's and so lay claim to at least 300 years of progress. Neither could I find the names of any very outstanding physicians in Vermont in the 1700's; and may I modestly suggest that the same may be true in the 1800's and 1900's also. Nevertheless I found much of interest to me in Vermont's Early Medical History, but it is so closely interwoven with her political history and geographical location that I am going to tell you a bit about that at the start.

Until after the close of the French and Indian War in 1763 Vermont was not a safe place in which to live, and few were the settlers that dared to clear farms within the present boundaries of the state; for raiding parties of Indians, using the Connecticut river and Lake Champlain as highways to lead them to the more populous centers of Massachusetts, New Hampshire and New York, scalped and burned as they went, wiping out small settlements, or driving the pioneers back to fortified places farther south.

Then, too, her territory was claimed by two different colonies, New Hampshire and New York, and Governor Benning Wentworth of New Hampshire on the one side and Governor Clinton and others of New York on the other,

*President's Address at the Annual Meeting of the New England Surgical Society at Boston, September 29, 1933.

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each sold townships regardless of the other, so that frequently settlers with grants from New Hampshire arrived to find the land already occupied by other settlers who had purchased the same from the New York governor, and vice versa. The conflicts that arose became serious and resulted in the formation of the Green Mountain Boys, military companies whose business it was to resist the encroachment of the New York settlers upon lands granted by New Hampshire. Thus early did Vermont identify herself with New England against New York. Even down to and through the War of the Revolution it is said that the Vermonters hated the Yorkers even more than they hated the British.

This controversy over the ownership of her land prevented the Continental Congress from recognizing Vermont as a separate state, for the powerful colony of New York claimed all this region and Congress dared not antagonize New York by recognizing Vermont. So from 1777 to 1791 Vermont stood alone, and between those years she was an independent nation, owing allegiance to no one, threatened on the north by the British army in Canada, bounded on the east and south by colonies that did not dare to support her request for inclusion in the Federation of States, because New York on the west opposed. And so for fourteen years Vermont ruled herself. She elected her own Committee of Safety, Governor and legislatures, levied taxes, coined money, maintained post offices and post roads, had a postmaster general, a supreme court, a standing army, and carried on diplomatic relations with the Continental Congress on the one side and the British in Canada on the other. So well and so craftily did she do all this that she was suspected of being disloyal to the Colonies and of planning to become a royal province, like Canada, but independent of her. However, the result was that she kept the English Army in Canada inactive for three years, they thinking that Vermont was about to announce her allegiance to the King and so should not be invaded. Nothing but the highest diplomacy—call it cunning or deceit if you will—protected this little state for years from being overrun by the British. She was exposed, helpless, denied aid by the Federal Government, and had to depend upon craft to accomplish what she could not do by force. All this was after she had played so important a part in the capture of Forts Ticonderoga and Crown Point and in the invasion of Canada at the beginning of the Revolution, and had done her share (under General Stark of New Hampshire) at Hubbardton and Bennington in 1777, to make Burgoyne's defeat at Saratoga possible.

I should have thought that a State continually at war with Indians, Yorkers, and British, to say nothing of the risks of ordinary living in a wild and unbroken wilderness, woud have needed doctors to sew up scalp wounds at least, but I find the name of hardly one physician among the original grantees of the several townships. No doubt there was an occasional army surgeon who, having come to Lake Champlain under General Amherst in the French and Indian wars, decided to remain and practice his profession when not busy farming, but certain it is that not until well down in the 1800's was any doctor able to earn his living by the practice of his profession alone, without some other means of support. Indeed, I find mention made regarding one early physician that he "confined himself strictly to the practice of medicine", as if that were an unusual thing. Many of them were farmers, most of them at one time or another in their careers went to the State Legislature, and others were civil magistrates of some sort. Several were preachers. Perhaps the craze to own land, which was so prevalent at that time, and which led most of the early settlers up into the wilderness, affected the doctors. It was said that no Vermonter could admire a landscape unless he owned it.

The names of the early Vermont doctors are preserved to us largely by their political connections, and little or nothing is said about their professional ability. For example Dr. Jonas Fay, the son of Stephen Fay who kept the famous Catamount Tavern in Bennington, the "Cradle of Vermont Liberty," was, to be sure, Surgeon under Ethan Allen at the capture of Fort Ti in 1775 and was continued in that office by the Massachusetts Committee of Safety. So he must have been something of a physician. But his name appears repeatedly as clerk of the various conventions from 1774 down, and also as a member of important committees, often as chairman. He was clerk of the General Convention held in Dorset, September 25, 1776, when the Independence of Vermont was first seriously discussed. And when final action on this was taken, January 15, 1777, the "Declaration and Petition of the Inhabitants of the New Hampshire Grants to the Continental Congress, announcing the District to be a Free and Independent State" was signed by four delegates, two of whom, Jonas Fay and Reuben Jones, were doctors.

This Dr. Reuben Jones, from Rockingham (in the southeast corner of Vermont) was clerk of one of the Conventions in 1774, and he it was who, on March 13, 1775 after the Westminster Massacre, had ridden "express and hatless to Dummerston to arouse the people." Probably he had the best saddle horse in town, and perhaps owned no hat. This Westminster Massacre was the first blood shed in the Revolutionary War.

Dr. Josiah Fuller was in Bennington, from 1762 to 1806. Although not an M.D. he followed Dr. Jonas Fay as surgeon at Fort Ti after its capture by Allen in 1775.

Dr. Benjamin Warner, father of Colonel Seth

Warner, the most noted of the Green Mountain Boys next to Ethan Allen, lived in Bennington from 1765-70, and Colonel Seth Warner himself was noted as a medical botanist and undoubtedly practiced in his regiment, much as sea captains to this day minister to the ills of their crews.

Another of the early physicians was Dr. Stephen Powers. He was one of the first settlers of Woodstock (and grandfather of Hiram Powers, the famous sculptor). He helped dress wounds at the battle of Bunker Hill, and always wore buckskin trousers, upon which he sharpened his scalpel. Many times in winter his practice led him on snowshoes through the woods, guided only by blazed trees.

Dr. Silas Hodges of Clarendon, Vermont was a surgeon in the Continental army and a member of Washington's military family.

Dr. Frederick Aubrey of Bradford had been a surgeon in the British army and is said to have dressed the wounds of General Wolfe at Quebec. He was apt to overcharge and once brought in a bill of $100 for removing a lot of flies from a boy's ear, claiming that he had saved the boy's life, which was worth more than $100. The boy's father thereupon presented his bill for two bushels of wheat at $50 per bushel, and the doctor gravely said, "I will dispute no man's account, we will pass receipts."

Dr. Roebeck of Grand Isle was a surgeon in the German contingent of the British army at the battles of Hubbardton and Bennington. After the Revolution he returned to Vermont and settled in Grand Isle. Not having money to buy drugs he sought them in the fields. He is quoted as saying, "Indian hemp is good for dropsy, spigot root for internal bruises, the bark of the red willow a sure remedy for fever and ague, burdock root with black cherry and white ash bark steeped in cider the very best remedy for spring jaundice."

Many are the stories told of the privations and narrow escapes of these early physicians. Dr. Adam Johnson of Danby, coming home on horseback late one night, was attacked by wolves, and finding them closing in on his horse, he threw his saddle bags among them to divert them. He afterward remarked that "those bags saved life that night if never before."

In August, 1814, in Brownington, a smuggler was shot in the leg. He was carried to the nearest house and Dr. F. W. Adams amputated, using a beech withe for a tourniquet and a razor and a sash-saw as his only instruments. Fifteen years later Dr. Adams is reported to have amputated above the knee four times in a single year.

These men are typical of the members of the medical profession in the pioneer days in Vermont. Although undoubtedly sadly lacking in medical knowledge, they were courageous and resourceful and their skill, perhaps, compared fairly well with that of physicians in other states at that time.

Apparently most of the obstetrics was handled by midwives. The first child born in Hartland was ushered into the world by a midwife, whom the father had drawn upon a sled from Charlestown, twenty-four miles on the ice of the Connecticut river. Mrs. Whitmore of Marlborough, wife of an early settler, acted as nurse and midwife. She officiated at more than two thousand births, and never lost a mother, and Mrs. Richard Wallace of Thetford acted as midwife in the seven surrounding towns, was present at 1666 births and is said never to have lost a mother of whom she had had charge.

Coming down to later times, in 1840 Dr. Luther Moseley of Arlington was the first to use adhesive plaster straps for extension in fractured femur. This method was copied by his relative Dr. James A. Gregg of New Hampshire and was later seen by Dr. Dixi Crosby, who wrote a paper upon the method for the New Hampshire State Medical Society, claiming that invention as his own, and for years it was called "Crosby's method." About 1859 Dr. Jonathan Allen of Middlebury introduced veratrum viride in the treatment of pneumonia, diphtheria, and rheumatism.

In Vermont, apparently at first, doctors were born not made. There were no laws limiting the practice of medicine until 1876 except that from 1820 to 1838 the right to collect fees was regulated. Anyone might practice if he could find people willing to take his medicines and advice and of our earliest physicians I can find very few who had the degree of M.D. Even so we were not much worse off than our neighbors in the rest of New England. For the practice of medicine was at a low ebb in the late 1700's in all of the colonies with the possible exception of Pennsylvania. Before that time it appears that quite a number of doctors educated abroad came to the colonies, practiced, and taught students, who, in their turn, practiced and taught other students etc. so that the medical degree of the earlier physician had become sadly diluted as generation followed generation with preceptor instruction only. That certainly is what happened in Vermont until the foundation of our first medical college in 1818, and none of the founders of that medical school held the degree of M.D. But more about the medical schools later.

Vermont's first effort to improve medical ed ucation in the state was the founding of so-called medical societies. These were chartered by the State with the right to hold property and to admit desirable men to membership, but with no right to exclude from practice those doctors who were not members. The first of these medical societies called "The First Medical Society of Vermont" was chartered in 1784, while Vermont was still an independent republic. It included practically all the physicians in Ben-

nington and Rutland counties, i.e., the southwestern part of the state. "The Second Medical Society in Vermont" was chartered ten years later, with similar rights and aims, in the county of Windham (southeastern) and "The Third Medical Society in Vermont" was founded in 1804, again ten years later, in Franklin county, the northern part of the state. These three societies were later (in 1813) merged with medical societies in several other counties, into the Vermont Medical Society, which for a time had a vigorous life but with the founding and rise of the three medical schools in Vermont, the State Society languished. None of these societies had the right to grant the degree of M.D., so that their power and prestige rested in the fact that they had libraries, instruments and apparatus to which their members had access and there was also the advantage of close association with the men who were the recognized leaders of the profession in the state.

It is evident, however, that certain other states recognized these medical societies, for at least one "diploma" from the First Medical Society has an acknowledgment on the back from the state of New York, dated August 26, 1806 (Saratoga County, Clerk's Office) which apparently had been registered to prove the right of the holder to practice in New York State.

In 1810, there were only eight medical schools in the United States, six of which were affiliated with colleges of Arts and Science, viz., University of Pennsylvania, 1765; Columbia, 1767; Harvard, 1782; Queens, 1792; Dartmouth, 1798; and Transylvania, 1799; and two were organized under the auspices of medical societies, viz., Physicians and Surgeons, New York, 1805; and Maryland Medical College, 1807.

In 1810, Middlebury College tried to get Dr. Nathan Smith away from Dartmouth to found a medical department in Middlebury. Dr. Smith was the most popular medical teacher in the country, and Dartmouth had more medical students than Harvard at that time. Dr. Nathan Smith was born in Massachusetts but spent his early life in Vermont and received his first medical education under Dr. Goodhue of Putney, Vermont. He also served with the Vermont troops in the Revolutionary War, and it was an operation which he witnessed in Vermont which led him to take up medicine. But Dr. Smith was dissatisfied at Hanover because they would not give him assistance in his teaching or a building for the Medical Department. For thirteen years he had been the entire medical faculty, and had given all the instruction except for a little help in three different sessions, and all for the salary of two hundred dollars a year. We must remember, however, that in 1810 Harvard's entire medical faculty numbered just three men, so that Dr. Oliver Wendell Holmes' joke that Dr. Smith occupied not a chair but a whole settee, while true, did not show a condition very different from that which prevailed in many medical schools at that time. Middlebury offered Dr. Smith two hundred and twenty dollars, and a lecture room in the college, and agreed to try to get some suitable person to be elected as professor of chemistry. But Dartmouth countered by appointing a professor of anatomy (Cyrus Perkins, M.D.) and erecting a medical building, for which Dr. Smith gave one acre of land, and his medical museum. This ended Middlebury's efforts to start a medical department at that time. Dr. Nathan Smith later founded the medical schools of Yale and Bowdoin and gave four courses of lectures at the University of Vermont.

In February, 1818, three men in Castleton, Vermont, established the first medical school in Vermont (Castleton Medical Academy). No one of these founders was a graduate in course in either Arts or Medicine, but all had been successful private teachers for some time and were men of great force of character and high reputation in the state. This school was incorporated by the state after it had given one course of lectures (14 weeks) and the next year, October 27, 1819, the charter was amended to allow the school to grant the usual degrees.

Nevertheless, since most medical schools were connected with colleges of Arts and Sciences, the Castleton Medical Academy united with Middlebury College in 1820, and so continued for seven years. From 1823-1826 its attendance was greater than that of any other New England Medical School. In 1838-39 there were no sessions, but late in 1839 the school was reorganized and it did not finally close until 1861. It had graduated over 350 physicians. Dr. William Tulley (Tulley powder) was professor of Theory and Practice and Medical Jurisprudence (1824-1839). The school gave an annual course of fourteen weeks' instruction and the fees were $55 for all lectures and a graduating fee of $16.

Also a summer course on Anatomy, Physiology, Botany, Materia Medica, and Chemistry and a fall and winter course on Anatomy and Operative Surgery were given. The catalogue naïvely says: "It is especially the design of this institution to offer facilities and means so ample for the acquirement of a thorough knowledge of anatomy that country students shall not be compelled to resort to the cities, at an increased pecuniary expenditure and the exposure of health and morals."

The Hubbardton raid upon the college in 1830, in which angry farmers marched upon Castleton Medical College to rescue the body of the wife of one of their number, which had been raised from a new made grave for dissection purposes, is indicative of the difficulty which medical colleges had in securing anatomical material. And this was by no means an isolated incident, for in the early days of the Woodstock school unwarranted disinterments became so common that the faculty finally had to prom-

ise not to use or allow to be used any body which had been disinterred in the state of Vermont. Perhaps New Hampshire, which was near by, suffered in consequence.

The medical department of the University of Vermont was the outgrowth of private classes taught by Dr. John Pomeroy, in Burlington, from 1810 to 1823. He was occasionally assisted by other physicians in the village and the classes grew so large that in 1814 he was forced to hire an empty store in which to hold the classes, which had formerly met in his office. He was a daring and skillful surgeon, and upon one occasion performed double amputation in the lower third of the femurs upon a patient who had been pronounced incurable by the associated physicians, only one of whom consented to be present at the operation and even he refused to assist. In spite of no anesthetic, and no asepsis, the patient made a complete recovery. At another time Dr. Pomeroy was called to attend a case of edema of the glottis and regardless of violent resistance on the part of friends of the patient he performed a tracheotomy, inserting a goose quill as a tube. This patient recovered. He had never seen a tracheotomy done and was threatened with lynching should the patient die. These two cases give a picture of the man. He was a friend of Dr. Beaumont, who at that time was stationed in Plattsburgh, N. Y. as an army surgeon, and frequently came to Vermont, being much interested in a young lady who lived in St. Albans.

John N. Pomeroy, his son, was a chemist, and not a doctor, but in 1816 he delivered a course of sixteen lectures on chemistry to his father's students, which is believed to be the first regular course of lectures upon chemistry ever given in this country to a medical class. His chemical experiments were attended by many of the townspeople. Upon one occasion when he had given notice that he had manufactured some nitrous oxide gas and would administer it to one of his students, he had a large audience of the townspeople, but at the last moment the student refused to be anesthetized. Nothing daunted, Mr. Pomeroy decided to take the gas himself and explained to the student how to give it. When the stage of excitement was reached Mr. Pomeroy overpowered the student who was giving the gas and forced him under the benches. He was loudly applauded by those of his audience who had the courage to remain through the experiment, and one young lady was so impressed by the whole affair that she soon after married Mr. Pomeroy, who was wont to say in after years "the success of the lecture might be open to doubt, but the certainty of its results in securing an introduction to the lady who subsequently became my wife is apparent to anyone." Neither Dr. Pomeroy nor his son ever received any pay for their teaching, but the medical class at the close of one course presented Mr. Pomeroy with a backgammon board, which he used for seventy years.

The Medical Department of the University of Vermont was regularly organized in 1823 with five professors, of whom Dr. Pomeroy was one and Dr. Nathan R. Smith, son of the great Nathan Smith, another. The college, after the first few years, was not very successful, and in 1836 closed for lack of students. It was not until 1854, that, largely due to the efforts of Dr. S. W. Thayer (the father of Dr. Charles Thayer, one time dean of Tufts Medical School), the school reopened. It is now the only medical school in Vermont.

It has been interesting to me to note what great stress is laid (in early accounts) upon a Medical Museum; for my own recollection of the Medical Museum of the University of Vermont is certainly not that it was of any great practical service in teaching, aside from the skeletons. A Medical Library was an entirely different matter, for in 1820 medical books were few and access to them was a great privilege. Since progress in medical science was very slow at that time, a book twenty-five and even fifty years old, might well be considered good authority.

The third medical school in Vermont was founded in 1827 at Woodstock, Vermont, by Dr. Joseph A. Gallup, who had previously been a professor and president of the Castleton Medical School. There were four members of the faculty. It affiliated with the Waterville College in Maine at first, and the degrees were granted by that Institution up to 1832, and from 1833 to 1837 the school was affiliated with Middlebury College, and degrees were granted by that institution. Two terms of thirteen weeks each were necessary to graduate. The school was prosperous largely because of certain distinguished men on the faculty—Willard Parker, Alonzo Clark and John C. Dalton, names to conjure with in those days. The school closed in 1856. At one time, owing to local jealousy, there were two medical colleges in Woodstock, and it was jestingly said that only Philadelphia could rival Woodstock in that respect.

As was perhaps natural, epidemic diseases excited the greatest interest if one may judge by the various medical writings of that time, and epidemic diseases were very prevalent in Vermont in the early days. Smallpox is first mentioned as having appeared in Bennington in 1773, and a town meeting was called which voted "not to give liberty to inoculate for the small pox." You all know of the great controversy regarding smallpox inoculation at that time, both in England and America. This vote seems to have been the first attempt at Public Health legislation in Vermont, and to this day Vermont ranks high among the states in her

Public Health laws. In 1856, she passed a law requiring the registration of births, deaths and marriages, which was one of the earliest registration laws passed in the United States. She was the first to have a compulsory school for health officers, paid for out of state funds and one of the first to maintain a State Board of Health Laboratory. Her method of management of anterior poliomyelitis cases has been studied and copied by many other states, and several foreign countries. But to go back. In 1776, smallpox was very prevalent in the American Army in Vermont and Canada, and many of the garrison of Fort Ti were stricken. The ill-fated expedition to capture Quebec in this year was defeated as much by the outbreak of smallpox in the American Army as by the force of the British arms.

In 1777 a pest house was established in Bennington and inoculations were allowed. It was also voted at a Bennington town meeting "that any person who should presume to have the infection on either of the several main roads through the town should be liable to a penalty of twenty pounds." In 1783 a state law was passed to prevent the spread of the smallpox, apparently the first piece of state health legislation to be passed by Vermont while she was an independent nation. For many years smallpox was the only quarantinable disease. In November, 1800, Zachariah Harwood, who, though not a regular physician, was believed to have peculiar skill in the management of smallpox, was licensed to inoculate, and though several hundred persons were inoculated, only one or two deaths occurred. In this same year, December 17, 1800, Dr. Benjamin Robinson, a young physician, advertised in the Vermont Gazette that he was "inoculating for the Kine, or as it is commonly called, the Cow Pox", and stating that he had the best European authority for warranting him in publicly declaring that "when a person has once had the Kine Pox he is forever after infallibly secure against catching the smallpox by any possible exposure." And a few months later, he advertised that three lads between thirteen and seventeen, who had had kine pox, went to the pest house and were actually inoculated by Mr. Harwood, and that not one of them was in the least degree affected with the smallpox. Since Jenner's report of vaccination was not published until 1799, it seems strange that it should have been used in Vermont in 1800. Dr. Gallup, in his sketches of "Epidemic Diseases" in the state of Vermont (1815) does not mention smallpox from which we may infer that inoculation and vaccination had so reduced the number of cases that in comparison with the others mentioned, he considered it of minor importance. This book was widely read. Upon the fly leaf of one copy which I have seen is written, "This work was republished in London in 1816, and then said to be the first American medical work ever republished in England."

According to Dr. Gallup, the epidemic diseases in Vermont in the early days, besides smallpox, were "spotted fever", "epidemic peri-pneumony", "dysentery", "angina epidemica", and "typhus epidemicus", as well as "pulmonary consumption". No doubt several of these names did not describe entities, and probably cases of several different diseases are often included under one diagnosis. Spotted fever, for example, indubitably sometimes was cerebrospinal meningitis, and sometimes tuberculous meningitis, for I find records of several autopsies (or dissections, as they were called) which mention meningitic adhesions and congestion, increased amount of cerebrospinal fluid, and often pus in considerable amounts, in the brain or cord. Several of the cases died in less than twenty-four hours from the first symptoms. Others lived several weeks and had relapses. One had "half a gill of pure pus in the substance of the left side of the cerebellum extending down to the great foramen and had been ill four weeks." Out of six cases reported, with autopsies, five were probably tuberculous meningitis and the sixth cerebrospinal meningitis. I am much amazed at the number of autopsies reported (1813).

As to treatment, the patients were bled and bled profusely and several times, often until they fainted. Opium is spoken of as a "high stimulant", along with "bark", "spirits", "cordials" etc. In fact, Dr. Gallup's book seems to have been written largely as propaganda for "bleeding". He lauds its value very highly and is scornful of those physicians who do not draw blood, or do so only in a half-hearted way. In this he and Dr. William Tully were at variance, and they had many spirited controversies. In his discussion of one disease Dr. Gallup covers several pages differentiating two varying types of that malady, but insists upon full bleedings in each form, regardless of whether it were of the plethoric or anemic type. He bled freely in pulmonary consumption even. "Epidemic peri-pneumony" is evidently what we call influenza, with cases of pneumonia, empyema, pleurisy and at least one case of laryngeal diphtheria. In this disease, when stimulants seemed indicated, Dr. Gallup advises a half ounce of brandy every four to six minutes, mixed with about fifteen grains of Peruvian bark and a little allspice and loaf sugar. No limit seems to be put upon the number of doses, but I should think that a half ounce of brandy with fifteen grains of quinine every four minutes would do something to a patient within an hour or so.

In five months of the year 1813, during this epidemic, 6,400 deaths occurred out of a population of 217,913 people. It would seem either that the disease was very virulent or that "bleeding" was not a very successful treatment.

Angina epidemica is evidently sometimes scarlatina and sometimes diphtheria. Dr. Gallup considered many strange things as causative of epidemics. He speaks of:

1. "The conjunction of the planets.
2. The general pestilential constitution of the air.
3. The general morbid state of the atmosphere.
4. Miasmatic effluvia.
5. Pestilential state of air (mephitis)".

He writes "Pestilential diseases spring from vapors evolved from the earth," and to prove it he records the deaths of an entire family in one night (evidently due to carbon monoxide poisoning from a smouldering bon-fire near the cellar wall) but the Doctor writes "the vapors were so powerful that lighted candles were instantly extinguished in the cellar." He also writes, "The spontaneous decomposition of animal bodies cannot be accounted for on any other principle than that of a surcharge of electron"—whatever that means.

Rudyard Kipling, who lived in Vermont for some years, is reported to have said at a doctors' banquet in London, that he had seen doctors in Vermont manage a practice "twenty miles in each direction, driving through eight feet of snow to attend an operation, digging the horses out of drifts and proceeding" and that it was one of the proudest things in his life to have been associated with the "real fighting men of this class".

Throughout these accounts I am struck by the outstanding characteristics of independence in the early settlers, and fearlessness and ingenuity in carrying out what they thought best. These characteristics still obtain. We Vermonters hate to be told what we must do and how we must do it. In recent years, when a certain authority, by a stroke of the pen tried to wipe out, along with many others, Vermont's only medical school, Vermont's reply was to tighten her belt and raise the medical teaching to conform with the required standard of a Grade A medical school. And if I read the temper of our doctors correctly, I predict that State Medicine, or the making of our physicians the hired help of any lay organization, be it a Bureau in Washington, or an Insurance Company in New York will be met with the same response that was given to the Yorkers who tried to take our lands. The Green Mountain Boys may not apply the "Beech Seal" to the interlopers as their ancestors did, but I trust them to find some way to preserve their professional dignity and personal independence.

MEDICAL SPECIALIZING DOOMED

According to a dispatch in the *New York Times* the general practitioner is to assume his former prominent position in the medical world according to Dr. G. Donald Guthrie of Sayre, newly-inducted president of the Pennsylvania Medical Society.

Dr. Guthrie said that the attitude of medical school graduates had shifted from postgraduate work toward general practice and that "the success of many of these young men has been most gratifying". He further declared that "as the army of poorly trained, inexperienced young specialists dwindles, the well-trained, alert, well-informed general practitioner will fill the gap — a group made up of men who can correctly diagnose and successfully treat 80 to 85 per cent of human ills without the help of the specialist or the modern instruments of the laboratory". Dr. Guthrie issued the warning that the physician's economic burden was in some cases almost ruining him and advised the Society that the care of the indigent should no longer be the medical profession's own personal charity.

THE PAYMENT FOR ACCIDENT CASES

Word has come to the *Journal* office that physicians are finding that the Industrial Act Board of Massachusetts is becoming less liberal in the approval of charges for industrial act cases.

Although the law provides that in exceptional cases the charges may run beyond those of the first two weeks' attendance, there has seemed to be a purpose to restrict the payment of physicians' bills to the two weeks' period, rather than allow the physician to continue treatment of a case so long as it may be required.

One physician has expressed the feeling that an effort is being made to divert certain cases to the care of physicians who are not selected by the patient.

THE TAXATION OF HOSPITALS

A movement is reported to be on foot in New York City, under the present administration, to end the tax exemption hitherto in vogue in dealing with hospitals.

The impression among medical men is to the effect that instead of having to pay taxes to the city, the hospitals should receive additional support from tax funds to supplement the contributions by charitably disposed persons.

Emphasis was placed on the importance of employment of federal relief funds in the care of the indigent in order to relieve physicians of the assumed responsibility so common heretofore of serving these people without pay.

VERMONT STATE MEDICAL SOCIETY

CESAREAN SECTION*

BY H. M. LITTLE, M.D.†

WHEN birth by the natural passage is impossible, suprapubic delivery by incision of the abdominal wall and the uterus offers, or should offer, certainty for securing a live child, with no more risk to the mother than that of an ordinary abdominal operation. The very safety of this procedure in competent hands has led to such abuse that it might be now described as the last resort of a competent obstetrician and the only resource of a consultant who is not a trained obstetrician. While done primarily for the sake of securing a live child, this desire should not cause the safety of the mother to be overlooked, but no other simple abdominal operation carries such high mortality as does cesarean section. While this might be excusable to a certain extent were all the children saved, the operation has been particularly discredited by the extremely high percentage of failures when maternal and foetal mortality are added together, as must be the case in determining the value of the procedure.

I have before me figures lent by my friend Professor Rudolph Holmes of Chicago, who wrote to some two hundred and fifty-nine (259) hospitals in this country to obtain statistics with reference to cesarean section. Two questions were asked:

1.—How many cesarean sections were done in your hospital in the past year?

2.—How many mothers died?

TABLE I

Hospitals	Cases	Total	Deaths	%
4	1	4	3	75
9	2	18	5	27.9
5	3	15	1	6.6
11	4	44	1	2.3
10	5	50	2	4
7	6	42	1	2.5
13	7	91	13	14
9	8	72	11	15
4	9	36	5	14
8	10	80	10	12.5
5	11	55	0	
3	12	36	3	8.3
2	13	26	3	11
3	14	42	3	7
23	17/27	496	40	8
9	30/50	359	18	5
5	50/100	320	9	3
2	over 100	309	17	5.2
		2095	145	6.92

From one hundred and thirty-three (133) hospitals reporting 2,095 sections, 145 deaths were reported, a percentage of 6.92, roughly seven per cent. On the other hand one hundred and twenty-six (126) hospitals reported 1,207 sections, but failed to report the number of deaths. Certain features of even this meagre report are worth consideration. Four hospitals reported each one cesarean and of these four, three died, a maternal mortality of 75 per cent. Where the operation was done a little more frequently the mortality rate was lower, but even when seven, eight, nine and ten sections per year were done, there was twelve to fourteen per cent maternal mortality. It is noteworthy that five hospitals reporting between 50 and 100 sections in the year had a mortality below three per cent, but two reporting over 100 sections show an increase to five per cent.

This is not a question of chance. It is a question; first, whether the particular hospital was a suitable place for cesarean section; secondly, whether the man doing a section only occasionally is competent to deal by this method with a case admitted as an emergency.

TABLE II

Hospitals	Cases (2nd Series)	Total
14	1	14
16	2	32
10	3	30
6	4	24
14	5	70
9	6	54
7	7	49
7	8	56
3	9	27
3	10	30
5	11	55
2	12	24
2	13	26
7	14	98
2	15	30
3	16	48
9	17/27	198
5	30/50	180
2	50/100	162
126		1207

In the second series of hospitals where no deaths are reported, we find 14 hospitals where one section was done in the course of the year. It would be interesting to know whether these cases fared more favourably than in the other series where one case per year carried a mortality of 75 per cent.

The outlook then for the mother, is by no

*Read at the Annual Meeting of the Vermont State Medical Society at Burlington, October 7, 1932.

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means good. Holmes, unfortunately, secured no statistics so far as the babies were concerned, but during the past few years statistics have been collected by a number of men in which, not only were the maternal deaths reported but also the foetal deaths.

Welz of Detroit—1926—in 154 sections, had 20 maternal deaths—13%, but also 17 foetal deaths—11%, a total of 24%.

Miller of Hartford, Conn.—1927—reported 444 sections with 20 maternal and 68 foetal deaths—4.5% and 15.3% respectively, a total of 19.8%.

Gordon of Brooklyn—1926—1801 cases, 130 maternal and 163 foetal deaths, 7.2 and 8.9 respectively, a total of 16%.

Miller of New Orleans—1926—291 sections—47 maternal 16.1% and 55 foetal deaths 18.9%—35%.

Sackett—New York Lying-In—578 cases, 25 maternal deaths—4.32%—No record of foetal results.

Thompson—Los Angeles—1322—56 maternal, 107 foetal, 4.2% and 7.9%—12%,

TABLE III

Survey by	Cesarean Sections		Mat. Deaths	%	Foetal Deaths	% Total
1. Welz: Detroit, 1926	154	24%	30	13	17	11
2. Miller: Hartford Hospital, 1904-1927	444	19.8%	20	4.5	68	15
3. Gordon: Brooklyn, 1921-1926	1801	16%	130	7.2	163	8.94
4. Miller: New Orleans, 1921-1926	291	35%	47	16.1	55	18.9
5. Sackett: N. Y. Lying-In, 1928-1931	578		25	4.32		
6. Thompson: Los Angeles, 1923-1928	1322	12%	56	4.2	107	7.9
	4590	16.7%	308	6.49	410	10.2

or,

combining all the above figures there will be noted approximately 4600 sections, a maternal death rate of 6.49% and foetal rate of 10.2%, a total of 16.7%. It will be noted that in practically no instances were there fewer babies lost than mothers lost, though the operation was done primarily in the interests of securing a live child, and that the combined failures varied from 16 to 35 per cent. Though high, the maternal mortality was practically what was found in the investigation of Rudolph Holmes.

Let me repeat, either there is something wrong with the operation as done, or there is something wrong with the person doing the operation.

A brief history of the development of the procedure would appear to suggest that it is not the operation that is at fault. The average layman believes that cesarean section began with Julius Caesar. Not only is this not the case but it is doubtful whether Caesar, who wrote to his mother, and moreover had younger brothers, was delivered by section, for till many centuries later than the Caesars no mother is known to have survived the operation. True, in the days of Numa Pompilius, several centuries before Christ (700 B. C.) the codified Roman Law, the Lex Regia, provided for the removal of a live child from the uterus of a woman dying pregnant and undelivered. This persisted later as the Lex Caesaria hence cesarean section. This law is still part of the code of many of the European countries. So, too, the Babylonian Jews in the second century before Christ, described deliveries, the Kariyath Habetan or the Jotze Dofan, suprapubic delivery under circumstances similar to those known to the Romans, but it remained for one Jacob Nufer, a swine gelder of Switzerland, to open his wife's abdomen and secure a live child without fatality. This in 1527. The operation done professionally probably dates from 1610 when performed by Trautmann of Wittenberg. For a century and a half the operation was occasionally done, but owing to infection and haemorrhage the results were almost invariably fatal. Be it remembered the open uterus was dropped back into the abdomens after the extraction of the child. In the nineteenth century the work of Semmelweiss, Pasteur and Lister did much to limit the possibility of infection at operation, but the turning point in the technique of operation came in 1876 when Porro suggested suturing the emptied uterus into the abdominal wound and removing its upper infected portion, while in 1882 Saegner was the first to suggest suturing of the uterine wound. This is just 50 years ago and I who have seen silk, silkworm gut and catgut used in closing wounds of the open uterus am unfortunate not to have seen silver wires used, or might have claimed to have lived through the complete development of cesarean section.

Rapid advances in abdominal surgery made for further improvement in this operation, but for 20 years, indeed up to 1907, there was very little modification of the technique of Saegner except in so far as the abdominal wound was concerned. The approach varied in some instances, high, medium or low incisions were made, low incisions in the uterus or even transverse incisions in the fundus, for the purpose of preventing adhesions of the sewn uterus to the abdominal wall. Thus this trouble was more or less eliminated as was also the danger from haemorrhage. There remained the danger of infection. In 1907 Frank of Cologne suggested that the lower the incision in the uterus the less danger from infection. This is today universally accepted, though in the past

25 years, the type of operation has changed in many ways. Latzo suggested a lateral incision with entry of the lower uterine segment between the folds of the broad ligament. Veit, Fromme and Hirst suggested incising the parietal peritoneum and then peritoneum of the uterus suturing these and opening the uterus low transperitoneally, while Sellheim combined these various measures, insisting that the results were not so much due to the manner of approach as to the fact of the incision being in the lower uterine segment. Today unless familiar with the low segment operation, it is doubtful whether anyone should undertake cesarean section. True, one may go for a long time without needing other than the classical section, i.e., incision from symphysis to umbilicus and the usual incision through the uterus, starting at the uterovesical fold, but in the vast majority of consultation cases, rupture of the membranes alone or associated with repeated examinations, makes the field of operation infected, particularly in proportion to the time lapsed since the onset of labour, and in these instances the low incision offers the only possible safety for mother and child.

Let it be remembered that the operation is primarily for the sake of the child; the child must be alive and in good condition, and that "good condition" is not entirely a matter of heart action, but with the resources of today, a child proved normal so far as is possible by means of x-ray. What does it profit to obtain a live hydrocephalic child or a live monster at the expense of a capital operation on the mother? Adequate prenatal care has made it possible to eliminate many of the acute accidents of pregnancy. DeLee's statement that prenatal care has abolished eclampsia is probably overdrawn. Placenta praevia can rarely be diagnosed before haemorrhage occurs, but though these two complications of pregnancy furnish frequent indication for the operation, there are other methods of treatment almost as satisfactory, if not more satisfactory, and for the vast majority of cases with an absolute indication for section, the need for operation may be established long before the onset of labour. In recognized obstetrical clinics ablatio placentae, previous abdominal operations, pregnancy complicating heart disease or disease of the thyroid, may be indications for the operation, but, in the absence of complicating haemorrhage, operation may be done at a predetermined time. Phaneuf of Boston, who has probably performed as many sections by various methods as any operator in America, believes that the ideal method in cases of emergency is the low segment operation suggested by Munro Kerr and Hendry of Glasgow.

Here the abdomen is opened in the usual manner, the intestines packed back and the bladder freed by an incision extending from one round ligament to its fellow at the opposite side. By pushing down the bladder the lower uterine segment is exposed and a crescentic incision is made parallel to the uterovesical fold. This offers ready access to the head of the child and after child and placenta are delivered is easy to repair. The transverse incision in the lower segment is probably better than the longitudinal, which is rarely possible unless the patient is well advanced in labour and there can be little doubt that an incision extending beyond the lower segment offers less chance of satisfactory repair than one confined to the lower segment.

This, then, is the case of suprapubic delivery. Classical section if you will, done at a time of election, low segment incision if there is possibility of infection or if the patient has been in labour. Remember that apart from infection the convalescence of these patients is frequently stormy, acute dilatation of the stomach, paralytic ileus, and thrombophlebitis are all apt to occur, and unless sufficiently familiar with the technique to be sure of the delivery of a live child with more than a fair chance of success so far as the mother is concerned, the operation should not be undertaken.

ANTE NATAL CARE*

BY IVAN Y. PATRICK, M.D.†

Mr. President, Members of the Vermont State Medical Society, Ladies and Gentlemen:

INTRODUCTORY

IT is a very great pleasure, indeed, to be present today at your meeting in Burlington. I consider it a great compliment that we have been invited to come from Montreal to share in your program. May I express to you in these few words our sincere thanks and our very deep appreciation.

In the brief time at my disposal it is quite impossible to cover thoroughly the large subject of *Ante Natal Care.* I have, therefore, found it necessary to omit many things I should like to say in order to discuss those portions of the subject which seem to me most important.

Ante natal care has been generally practiced, with more or less thoroughness, for some time, yet it is not very old. Many of you can remember the time when few physicians were prepared to render this service to their patients and still fewer patients willing to accept it. Communities still exist in Canada, and I expect in your country as well, where prenatal care is unheard of.

*Read at the Annual Meeting of the Vermont State Medical Society at Burlington, October 7, 1932.

†Patrick—Associate in Obstetrics and Gynaecology, Royal Victoria Hospital, Montreal, Quebec. For record and address of author see "This Week's Issue," page 811.

Therefore it is still necessary to impress upon some members of the medical profession the great importance of this phase of obstetrics, and to educate the public to a realization of its value.

FIRST CONSULTATION

We begin ante natal care, then, by educating the woman to consult her physician as soon as she suspects herself of being pregnant and the doctor's serious responsibility commences with this first visit.

HISTORY

A history of the patient's past life, from birth onward, should be elicited, with care and thoroughness. So often the occurrence of previous illness, such as tonsillitis, rheumatic fever, chorea, diphtheria or scarlet fever, will guide the inquiring physician to unsuspected cardiac or renal disease. Even the family history is sometimes relevant, pointing to mental instability, tuberculosis or the probability of twin pregnancy.

PHYSICAL EXAMINATION

The physical examination at this first visit visit should be complete, special attention being paid to any system rendered suspect by the history. The bimanual examination is most important. It must be thoroughly and carefully carried out, particularly keeping in mind the possibility of extra-uterine gestation, pelvic tumors, incarcerated uterus and abnormalities of the genital tract. The interior of the bony pelvis is to be carefully palpated, the skilful and experienced hand frequently recognizes pelvic deformities which the pelvimeter fails to expose.

Pelvimetry. This is of great value, in that it reveals the majority of gross pelvic deformities, but one must realize that it has its limitations. Occasionally a deformed pelvis, which renders delivery difficult or impossible, may have all its ordinary measurements perfectly normal. The most striking example of this occurs in the obliquely contracted type. Again, a pelvis quite roomy enough for a small baby may be entirely inadequate for a large one. The pelvimeter is useful, but it must not be given an importance out of proportion to its value. The final decision must await the full development of the foetus during the last weeks of pregnancy.

GENERAL DISEASES

Diseases outside of the genital tract are frequently discovered at this first consultation, chronic cardiac, renal and pulmonary conditions are fairly common. In such cases, a grave responsibility rests upon the physician. He is called upon to determine whether pregnancy is to be terminated or allowed to continue; on the wisdom of his advice depends a life, often two lives. No one can lay down an inflexible rule to be followed in these cases, each one must be carefully studied and only judged after all the evidence is considered. One should endeavor to estimate the risk as accurately as possible, and then lay all the facts before the patient and her husband. I believe the woman, herself, should cast the deciding vote in a doubtful case. If pregnancy means a risk to her life and future health, she has the right to decide whether she will take it.

THE VOMITING OF PREGNANCY

This is usually the first difficulty encountered in the ante natal period. It is regarded by some authorities as a toxemia, by others as a neurosis and by still others as a combination of the two. The methods of treatment recommended are legion and none are specific. I confess to being one of those who look upon it as a toxemia with a large factor of neurosis. Successful treatment depends greatly upon the physician's skill in solving his patient's psychological make-up and offering the appropriate suggestions. These vary with the individual patient and time does not permit me to discuss them in detail. One may mention, however, that carefully disguised threats and promises of reward for improvement are two effective weapons. Useful aids to this treatment are fat-free diet, small meals at frequent intervals, seven in a day. Hospitalizing the patient, intravenous glucose, salines, and colonic irrigations are indicated. Endocrine preparations are frequently used, but their value is still in doubt.

MINOR POINTS

The primiparous woman is almost always profoundly ignorant of all things pertaining to pregnancy. She needs advice, guidance on her manner of living, encouragement in her discomfort and treatment for many minor ailments.

Constipation is almost invariably present and must be combated by all means at our disposal. ination in a toxic patient will frequently precipitate a convulsion.

It is a well-recognized fact that faulty elimination in a toxic patient will frequently precipitate a convulsion.

Diet should aim at correcting constipation, fruits and vegetables occupying a prominent place. Many women are inclined to overeat during pregnancy, as is witnessed by gains in weight of forty or fifty pounds. Such patients ought to be placed on a diet with a caloric intake suited to their needs. It is stated that during the War, in districts where food supplies

were short, eclampsia almost disappeared. This is a thought provoking observation.

Teeth should be cared for by a good dentist. It is important to maintain the mouth in a healthy state. The old dictum, "For every every baby, a tooth" may be proved false if the pregnancy is wisely managed and tooth decay treated as soon as it appears.

Cod liver oil is advocated and has unquestioned value, but in my experience few pregnant women are able to tolerate it without marked digestive disturbance. The highly concentrated preparations now on the market are taken with less difficulty, but one should be careful to keep the administration down to reasonable limits. It is conceivable that in our enthusiastic efforts to increase the absorption of calcium we may produce a hard foetal skull, incapable of molding during labor, and thus endanger the baby's life.

The question of sexual intercourse is frequently avoided, both by patient and physician, on account of the peculiarly delicate nature of the subject. This is a matter, however, on which it is most important to advise the patient. Intercourse shortly before the onset of labour, has many times resulted in serious puerperal infection. Premature labour and abortion can frequently be traced to this cause. Abstinence throughout pregnancy would be ideal, but is probably too much to expect of human nature. One may suggest, however, that intercourse be indulged in as seldom as possible, and be absolutely forbidden during the last two months.

Bathing shoud be frequent to aid elimination through the skin, the shower is preferred to the tub.

Clothing is to be loose and should hang from the shoulders. A corset or abdominal support, fitting snugly enough to take the weight, ought to be worn during the latter months. Circular garters aggravate varicose veins and are therefore are to be avoided.

Feet are frequently neglected. A shoe with medium low heel and good arch support should always be worn. The common and pernicious habit of wearing bedroom slippers is to be condemned.

Exercise should be regularly taken, but the more violent forms must be avoided.

FREQUENCY OF EXAMINATION

Examination and observation of the patient should take place at frequent intervals. The normal case should be seen by her physician every three weeks during the first six months, after that every two weeks. Anything abnormal may require more frequent visits.

THE TOXAEMIA OF PREGNANCY

This is always threatening and continues to hold the most prominent position in the prenatal period. I cannot enter into any thorough discussion of toxaemia, but I should like to emphasize one or two points which are frequently ignored.

Blood pressure readings are sometimes misinterpreted and for that reason a plain warning of impending danger is unrecognized. Many women during pregnancy have a low systolic pressure in the vicinity of 100 to 110. One must realize that this is their normal pressure. Consequently when the systolic rises to 120 or 130, it is evidence of a toxaemia, a fact that is very seldom recognized. This is the type of patient who will sometimes develop eclamptic convulsions with a systolic pressure below 140, often to the utter amazement of the attending physician. However, the warning is usually there if we have eyes to see it.

The diastolic pressure is even more valuable than the systolic as a warning of early toxemia. One can almost accept it as an axiom that a diastolic of 90 or over is to be regarded as a very serious sign.

It is well to realize that there are, occasionally, cases of what may be called fulminating toxaemia. They may develop to the most alarming severity in a few days. Fortunately they are rare and one has ample warning in most cases. To guard against this fulminating type, however, it is wise to inform every patient concerning the symptoms and signs of toxaemia. They should be instructed to report immediately on the appearance of edema of the face, visual disturbances, headache, vomiting or decreased urinary output.

Examination of the urine is so regularly carried out that it seems scarcely necessary to mention it. One might perhaps suggest that a microscopic examination for pus be part of the routine in order to recognize a symptomless pyelitis.

Pruritis Vulvae is a fairly common complaint. It is well to remember that many of these cases are due to trichomonas, which yields to local treatment with mercurochrome or picric acid.

Sacro-Iliac Pain is of frequent occurrence. It usually disappears at once when a strapping of adhesive plaster is applied.

THE LAST WEEKS

The last few weeks of pregnancy are important ones. The foetus is now fully grown and it is time to finally settle the question of its passage through the bony pelvis. The normal case with head deeply engaged offers no difficulty, the impossible pelvis is readily recognized and cesarean section is arranged for, it is the border-line case between these two which creates the difficulty. This is perhaps the greatest test of the obstetrician's skill and judgment. It is not sufficient to determine that the foetal head can be pushed or pulled through the pelvic canal by brute force, but one must try to determine if it will come through undamaged. In such cases, to accurately estimate the dan-

ger to the baby calls for skill and judgment of a very high order.

One should first determine if the head is engaged or if it can be passed through the pelvic brim. It is necessary to recognize that there is a difference between fixation and engagement. Because a head is fixed in the brim of the pelvis, it does not follow that it is engaged or that it will pass through the superior strait without damage. To be engaged the biparietal diameter must have passed through the plane of the brim. In doubtful cases Muller's test is most useful. With the patient under anesthesia, a hand on the abdomen forces the foetal head into the brim, while the fingers of the other hand in the vagina note the descent of the head. When the biparietal diameter enters the plane of the brim the most dependent part of the foetal head will be at the level of the ischial spines. If this maneuvre can be successfully accomplished, the head will pass, provided, of course, that there is no barrier at the outlet. This procedure should be followed in every doubtful case before the onset of labour. It is the most important piece of evidence we can elicit in deciding on which case to section, and which to put through labour.

In these last weeks of the ante natal period it is also of the greatest importance to recognize abnormalities of position and presentation. The diagnosis can, as a rule, be made more readily before the onset of labour than after.

If the breech, the occiput posterior, the poorly flexed head, the hydrocephalus or the transverse position are diagnosed early it is unlikely that the labour will be mismanaged. Their late recognition may lead to tragic results.

The value of auscultation of the foetal heart is too well known to require discussion here, but may I emphasize this one point which seems to be frequently forgotten. A persistently irregular foetal heart with an excess of amniotic fluid nearly always means developmental abnormality of the foetus such as spina bifida, hydrocephalus or an anencephalic monster.

Finally, the x-ray has earned a place in obstetrics. It is not to replace palpation or clinical diagnosis, but it has proved a definite aid in confirming or correcting the diagnosis of deformed pelvis, twin pregnancy, hydrocephalic and anencephalic monsters, disproportion and abnormalities of positions.

CONCLUSION

These are some of the things that go to make up ante natal care.

By frequent examination, by careful observation and by foresight we try to prevent rather than cure.

It is preventive medicine in the best sense of the term.

By these means we endeavour to guide the woman and her unborn child safely through the dangers of pregnancy.

MISCELLANY

VERMONT DEPARTMENT OF PUBLIC HEALTH

AUGUST, 1933

During the month of August, communicable diseases have been reported to this Department as follows: chicken pox 10, diphtheria 2, measles 14, mumps 21, scarlet fever 17, poliomyelitis 4, typhoid fever 2, undulant fever 1, tuberculosis 12 and whooping cough 43.

A total of 1,715 examinations were made by e Laboratory of Hygiene during August. These ex aminations are classified as follows:

Examinations for diphtheria bacilli	103
" " Widal reaction of typhoid fever	40
" " undulant fever	42
" " gonococci in pus	153
" " tubercle bacilli	160
" " evidence of syphilis	457
" of water, chemical and bacteriological	214
" " water, bacteriological	263
" " milk, market	189
" " milk, submitted for chemical only	11
" " milk, submitted for microscopical only	4
" " foods	7
" " drugs	0
" for the courts, autopsies	4
" " the courts, miscellaneous	20
" " evidence of malaria	2
" of animal heads for evidence of rabies	0
Miscellaneous examinations	46
Autopsies to complete death returns	0

Twenty-seven cases of gonorrhea were reported to the Division of Communicable Diseases, and twenty cases of syphilis. Two hundred Wassermann outfits and one hundred and ninety-two slides for gonorrhea were distributed.

The nurses of the Division for the After-Care of Poliomyelitis saw 59 patients in their homes, two at the office and 485 at clinics. Seven calls were made on nurses and overseers of the poor regarding the care of patients. Ten new pieces of apparatus were fitted, 11 braces repaired and 42 corrections made to shoes. Dr. Frank R. Ober and assistants, together with poliomyelitis after-care nurses, conducted clinics in ten towns of the state, with an attendance of 485 patients. The Vocational Worker of this Division reports $360 taken in during August.

The State Advisory Nurse visited eight towns this month, assisting in preschool clinics, home nursing meetings, and talks to Home Bureau Groups. Three hundred and thirty birth certificate acknowledgments and 262 pamphlets were distributed by this Division.

FRANKLIN COUNTY SOCIETY NOTES

Dr. H. H. Johnson of St. Albans who was operated on for appendicitis Monday, September 18, is doing well.

Dr. G. C. Berkley of St. Albans has been ill and confined to his home for more than a month past.

The widow of the late Dr. Sherwood died at the home of her daughter Dr. Grace W. Sherwood last week.

RECENT DEATHS

GIFFORD—John Pearl Gifford, M.D., Randolph, Vermont, was born in Randolph, Vermont, December 14, 1871, and died in a Boston hospital August 30, 1933.

Dr. Gifford graduated from Dartmouth Medical College in 1894 and was a member of the American College of Surgeons.

He began practicing in Randolph in 1897.

His widow, Mrs. Eliza Folson Gifford, is the only surviving relative.

CLARK—Frederick Ellsworth Clark, M.D., 64, a practicing physician of Burlington, Vermont, for many years, died suddenly September 11, 1933.

Born in Ashburnham, Mass., February 1, 1869, Dr. Clark attended Cushing Academy, from which he was graduated in 1888. In 1894 he was graduated from the University of Vermont College of Medicine and until two years ago was associate professor of pathology at the University. In 1916 he was commissioned as major in the Medical Corps of the United States Army and served for the duration of the World War.

Dr. Clark was a member of Washington Lodge, No. 3, A. F. and A. M., the Scottish Rite, and the Phi Chi medical fraternity.

Dr. Clark leaves his widow, Linnie Simonds Clark, two daughters, Linda of New Rochelle, N. Y., and Mrs. Paul D. Clark of Burlington, and two grandchildren.

ESSEX NORTH DISTRICT MEDICAL SOCIETY

The Ninety-Second Quarterly Meeting was held Wednesday, October 18, 1933, at Andover Academy.

PROGRAM

1. Business Meeting.
2. Dinner at Phillips Inn.
3. W. H. Robey, President of the Massachusetts Medical Society—The Affairs of the State Society.
4. Pitfalls in Surgical Diagnosis—Arthur W. Allen, Massachusetts General Hospital.
5. Pitfalls in Medical Diagnosis — B. H. Ragle, Massachusetts General Hospital.

Business Meeting and Scientific Program at Peabody House, across Main Street from the Campus.

Censors will meet at Hotel Bartlett, Main Street, Haverhill, at 4 P.M. Thursday, November 2, 1933. Candidates must present applications and show diplomas to the Secretary at least two weeks in advance.

F. H. Coffin, M.D., *President*,
E. S. Bagnall, M.D., *Secretary*,
281 Main Street,
Groveland, Mass.

Postgraduate Instruction Courses

Forty-seven Fellows from the Essex North District Medical Society have signed up. The selection of courses was based on the number desiring each course.

The first course, "Practical Medical Principles," was given by Dr. Chester M. Jones and Dr. Richard H. Miller on Friday, October 13, at 4:15 P.M. in the Lecture room of the Stephen Henry Gale Hospital, Haverhill.

"Gastro-Intestinal Disease (A)" will be given by Dr. Isaac R. Jankelson and Dr. Sara M. Jordan on Friday, October 20, at 4:15 P.M. in the Council Chamber, City Hall, Main Street, Haverhill. (*Note change in meeting place.*)

On October 27 at 4:15 P.M. "Cancer. Its Early Diagnosis and Treatment. Biopsy," will be given by Dr. Ernest M. Daland, Dr. Joe V. Meigs, Dr. Richard Dresser and Dr. Roger C. Graves in the Council Chamber, City Hall, Main Street, Haverhill.

Fees: Fee for the 10 courses is $5.00 payable at the first course attended.

Fellows who have not signed up for the instruction and desire so to do, may sign at the first meeting they attend.

The list of courses follows:

Vascular Disease
Arthritis
Medical and Surgical Emergencies
Syphilis and Dermatology
Practical Medical Principles
Pathological Pregnancy
Gastro-Intestinal Disease A
Vaccine Therapy
Diabetes and Vitamins
Cancer.

The courses will be given on successive Fridays and announcement of the subject and instructors given in the *Journal*.

F. W. Anthony, M.D., *Chairman*.
81 Merrimack Street, Haverhill.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 19421

ANEMIA OF UNSUSPECTED ORIGIN

MEDICAL DEPARTMENT

PRESENTATION OF CASE

DR. RICHARD B. CAPPS:* This is the case of a fifty-eight year old leather worker who entered the hospital complaining of weakness of six months' duration.

One year before admission he noticed some slight tingling in his fingers, also slight edema of the ankles in the evening. Six months before admission he noticed the onset of weakness and some dyspnea on exertion. These gradually increased. For three months he had had occasional numbness of the legs at night and some palpitation. A month before admission he began to bruise easily. A week later he began to have anorexia, epigastric soreness, and nausea and vomiting which had progressively increased until for three days before admission he had been vomiting everything he ate. He had lost twenty pounds in the six months before entry. His stools had been perfectly normal,—no tarry stools, no blood. He had noticed some involuntary twitching of his legs in bed.

Physical examination showed a well developed but rather poorly nourished man lying flat in bed in no distress. There was marked pallor. The chest was barrel shaped. Expiration was prolonged. The heart appeared normal on physical examination. The abdomen was rather tense and not easily palpated. No masses were made out. There was edema of the ankles.

His red cell count on admission was 2,500,000, with Tallqvist hemoglobin of 50 per cent. The cell volume index was found to be slightly above normal. The volume index was 1.2 and the color index 1.2. The smear showed an absence of achromia and some large well filled cells, but not sufficient variation from normal on which to make a diagnosis of pernicious anemia.

A gastro-intestinal series showed a gross variation in the shape of the stomach and deformity of the duodenal cap.

The patient was put on a diet and given tincture of belladonna, but the vomiting continued. After he had been in the ward for four or five days it was noted that he was running a large trace of albumin in the urine. The non-protein nitrogen was found to be 160 milligrams and the renal function zero. He developed hyperpnea and marked acidosis and died two weeks after admission in typical uremia.

*Recently senior interne on the East Medical service.

CLINICAL DISCUSSION

DR. CAPPS: He was admitted with a diagnosis by his own physician of pernicious anemia. It was felt that with a red cell level of 2,500,000 he was entirely too sick for pernicious anemia and probably had a carcinoma of the stomach. The X-ray Department made a diagnosis of duodenal ulcer, much to our surprise. This diagnosis was not entirely satisfactory, especially as there was no response to ulcer treatment. When the albumin was noted it was suggested by the resident that perhaps he had chronic nephritis. This diagnosis was soon confirmed, and explained satisfactorily all of the findings.

The discharge diagnosis was chronic nephritis and duodenal ulcer with secondary anemia.

DR. WYMAN RICHARDSON: I did not come on service until this package had been tied up for me, so I did not have to worry about diagnosis. I think from the history that the possibility of pernicious anemia was certainly justified and the history of tingling of the fingers, the gradually increasing pallor, the weakness, and also the vomiting might have been explained on that basis. As one saw the patient, however, and I might add, from later knowledge of the diagnosis, he was not quite of the type that usually does have pernicious anemia, not the usual florid type. When the x-ray diagnosis of ulcer appeared, that complicated the matter a good deal because it was thought then that the anemia might well be explained on the basis of a hidden and constant hemorrhage from the duodenal ulcer. Finally with the finding of a high non-protein nitrogen in association with urinary changes it seemed probable that the anemia could be explained on the basis of chronic nephritis.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Chronic nephritis.
Uremia.
Duodenal ulcer.
Secondary anemia.

ANATOMIC DIAGNOSES

Chronic vascular nephritis.
Arteriosclerosis, moderate aortic.
Prostatic hyperplasia.

DR. TRACY B. MALLORY: The diagnostic problem in this case was of course to prophesy what type of chronic nephritis he would show at autopsy. At fifty-eight years the statistical grounds for believing he had chronic vascular nephritis would make an almost overwhelming probability in favor of that. On the other hand his hypertension was not very marked, diastolic 86 and systolic 180. One would rather

expect a distinctly more elevated diastolic, at least, than that. The urinary findings were not very marked but were perfectly consistent with either diagnosis, vascular or glomerular nephritis.

The kidneys of chronic vascular nephritis are not generally very atrophic, that is, the pair are apt to weigh in the neighborhood of 150 grams rather than less and the general color of the external and cut surfaces is apt to be distinctly red rather than gray. At autopsy we were rather surprised to find a pair of kidneys that weighed only 100 grams and that in color were a cross between red and gray. On the whole the gross appearance of the kidneys was more like the end result of glomerular nephritis than of vascular.

The diagnosis that one would naturally make from the clinical history, in view of the patient's age, is confirmed by the histologic examination, although the gross appearance of the kidneys was not the usual one for nephrosclerosis.

A PHYSICIAN: How about the duodenal ulcer?

DR. MALLORY: There was none. The duodenum was entirely negative on very careful examination.

We were limited to an abdominal incision, so that I cannot say whether the heart was hypertrophied or not. I think there is very little doubt that it must have been.

DR. CAPPS: How about the bone marrow?

DR. MALLORY: We have not examined that as yet. I think it will undoubtedly be hyperplastic. It usually is in vascular nephritic cases.

DR. WILLIAM B. BREED: I think it would be interesting if we could have the x-ray diagnosis of duodenal ulcer demonstrated to us.

DR. AUBREY O. HAMPTON: This patient was examined twice. At the first examination no diagnosis was made. At the second examination it was thought the duodenum was deformed. As usual, we cannot tell much about the gastrointestinal tract from the films alone. This is a concave pressure defect. That is all I can say. These films do not prove or disprove that the duodenum was or was not deformed from intrinsic disease.

DR. CAPPS: I was told that the diagnosis was made on a constant deformity of the cap and that no crater had been demonstrated.

DR. HAMPTON: Ordinarily if the duodenum is deformed, no matter what it looks like, if you can rule out obvious extrinsic pressure defects such as gall bladder, spine and what not, you call it duodenal ulcer. I suppose it was thought that since the center of the concave defect showed this irregularity, it was not all due to pressure. Was the duodenum adherent to the gall bladder?

DR. MALLORY: No. There was nothing at all to suggest any cause for the x-ray appearances.

CASE 19422

CAN THE APPEARANCE OF THE KIDNEYS BE PREDICTED BEFORE AUTOPSY?

MEDICAL DEPARTMENT

PRESENTATION OF CASE

DR. CHARLES F. WARREN:* This case came to our Outpatient Department five years and five months before the boy's first admission to the wards. At that time he was ten years of age. He complained of enuresis, frequency and nervousness.

He was perfectly well as a child, except for measles, and blood poisoning after stepping on a nail, until a year before he came to the Outpatient Department, when he found he could not play like the rest of the children and was generally run down. He also had nocturia and occasional incontinence. He was found to be rather pale and slender, running a pulse of 100. The urine showed a large trace of albumin. The tonsils were rather large; a nose and throat consultant thought they were definitely infected. They were removed at that time. The diagnosis was thought to be possible orthostatic albuminuria.

We did not see him again for five years and five months. He then gave a history that he had never been really well during the five years, had not been able to keep up with his playmates, although he was able to go to school and do his work satisfactorily. Two months before entry he first noticed edema of the legs and puffiness around his eyes. He still had frequency ten times a day and three or four times at night.

First admission. He was found to be rather pale and pasty, with edema of the legs and face and some of the scrotum. The fundi showed a few hemorrhages. The aortic second sound was increased, louder than the pulmonic second. He had a systolic apical and basal murmur. The blood pressure was found to be 170/105.

Laboratory studies showed a normal red cell count, but a white cell count of 24,000 on entry which fell to 12,000 a week later. The urine showed a large trace of albumin, concentrated as high as 1.026. The non-protein nitrogen was 32 milligrams. The serum protein was 4, the cholesterol 337. The renal function showed only 6 per cent excretion of the dye in two hours and ten minutes, a 10 cubic centimeter specimen.

He was allowed to go home, where he stayed for four weeks.

Second admission. He returned at the end of that time with no edema of the extremities, but with ascites and effusion in both chests.

The laboratory findings were similar to those of the previous entry except that the non-pro-

*Recently senior interne on the West Medical service.

tein nitrogen had risen to the neighborhood of 60. The basal metabolic rate, taken several times, ranged from —29 to —35.

On a high protein diet the edema improved considerably. He was discharged with a very poor prognosis.

He came in for the last time four years later. He was then nineteen years old.

History of interval. For two years he had done much better than we had expected. He had even been able to play ball, and had gone about his school work very well. Two weeks before entry he suddenly began to cough, raising a small amount of whitish sputum. A week before entry he was troubled considerably with dyspnea and orthopnea, became nauseated and began to vomit. He had difficulty in seeing and had become drowsy. Two or three days before admission his parents noted that he had become twitchy and jumpy.

Third admission. We found him again to be pale and pasty, with a puffy face. The breath was uriniferous, the tongue dry and coated. He had marked hypertensive neuroretinopathy with many patches of fresh white exudate. The heart sounds were regular, the rate 120. There was a systolic murmur at the apex and the base. The blood pressure was 240/140. He showed considerable peripheral arteriosclerosis. He had an effusion of fluid in both chests, ascites and generalized abdominal tenderness with more marked tenderness in the flanks and the costovertebral angles. The reflexes were generally increased and there was occasional twitching of the muscles.

At this time there was no urine specimen, as he was anuric for two days in the ward. The white cell count was 17,600, the red cell count 2,110,000, the hemoglobin 45 per cent. The smear showed 90 per cent polymorphonuclears, 6 per cent lymphocytes, 4 per cent monocytes. The non-protein nitrogen was 92 milligrams. The carbon dioxide combining power was 51.2 volumes per cent, the serum protein 4.2.

The night of entry he had an attack of breathing that was typically asthmatic in nature and responded immediately to morphine. Following the morphia he never regained full consciousness. He became more and more drowsy, began to twitch more and more, went into coma and died.

Clinical Discussion

Dr. Warren: We made a diagnosis of chronic glomerular nephritis at the time of his first discharge and of probable nephrotic phase of glomerular nephritis at his second discharge. We thought he died of uremia and chiefly chronic glomerular nephritis; also we believed he had hypertensive heart disease and some degree of congestive failure.

Dr. Aubrey O. Hampton: I think the heart measurements are not going to be satisfactory, since I cannot see the left border because of what looks like fluid in the pleural cavity. The heart is not displaced to the right side so much as we should expect. As a matter of fact it is to the left because of rotation. This haziness might be a thin layer of fluid on the right. It does not obliterate the diaphragm or the costophrenic angles. The measurements are of no value. The sinuses seem normal.

I have been tempted to guess at the size of the kidneys many times, and lately we have been measuring them. The normal length is supposed to be about 12 centimeters, is it not? Since the intravenous pyelogram has been in vogue we have been wondering if we could make a diagnosis of contracted kidneys. This kidney measures 13 centimeters in length.

Clinical diagnoses (from hospital record)

Chronic glomerular nephritis.
Uremia.
Hypertension.
Hypertensive heart disease.

Anatomic diagnoses

Chronic glomerular nephritis.
Cardiac hypertrophy, hypertensive type.
Bilateral hydrothorax.
Ascites.
Pericardial effusion, hemorrhagic.

Pathologic Discussion

Dr. Tracy B. Mallory: This case was followed for a considerable period of years, including a first admission with albuminuria and nothing else. The second admission added marked edema and low serum protein, some red cells, but not much elevation of blood pressure, certainly sounding like a nephrosis superimposed on a nephritis. Eventually he goes on to marked hypertension and uremia, as we should expect in a case of chronic glomerular nephritis. From the clinical point of view I think one would be as safe in diagnosing chronic glomerular nephritis in this case as one ever could be.

Just before the autopsy one of the house officers came over and started to argue with me as to what the kidneys would look like at the autopsy table. He said we ought to find small red kidneys and I said they could not possibly be that, since chronic glomerular nephritis, which we both agreed was the diagnosis, characteristically results in a small white kidney. At autopsy we found that he had small red kidneys.

Microscopically, however, it is a perfectly typical glomerular nephritis. In other words, taking this case in connection with the one before, I think in each instance the clinical history and the microscopic findings on the kidneys agree very closely. The gross appearance of the kidneys was totally misleading in both cases. The statement that a chronic glomerular nephritis will give a small white kidney and chronic vascular nephrosis a less small,

red kidney is correct 80 to 85 per cent of the time; but the rule undoubtedly has exceptions, and the two cases that we have taken up so far today are both definite and complete exceptions.

Dr. William B. Breed: This case which has been under observation for so many years brings up the old question as to how often we are justified clinically in making the diagnosis of nephrosis alone, or whether we must say a nephrotic phase of chronic nephritis. How often do you find at the autopsy table kidneys about which you can say "This is just nephrosis"?

Dr. Mallory: Practically never here, chiefly because the Pediatric service in proportion to the rest of the hospital is a small one. In large hospitals for children I think there is no question that they do see cases which clinically and anatomically present the pure nephrotic picture without any trace of nephritic elements. There are many good men on both sides of the fence there, some claiming that the two are always mixed and others claiming that without any question pure nephrosis without any nephritic element does occur. Klemperer in New York is one of the latter, and there are a number of the Germans. I am personally inclined to agree that it does occur in children not very rarely, but rarely if ever in adults.

Dr. Wyman Richardson: The case is interesting also in regard to the question of albuminuria. I should like to know whether it was really orthostatic when first observed. In other words, was the specimen passed when the patient had been lying down all night negative for albumin or was it not?

Dr. Mallory: In view of the subsequent story I think one has a right to be very skeptical about the first diagnosis.

The New England
Journal of Medicine

SUCCESSOR TO

THE BOSTON MEDICAL AND SURGICAL JOURNAL

Established in 1828

Published by THE MASSACHUSETTS MEDICAL SOCIETY under the jurisdiction of the

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SUBSCRIPTION TERMS: *$6.00 per year in advance, postage paid for the United States, Canada $7.04 per year, $8.52 per year for all foreign countries belonging to the Postal Union.*

Material for early publication should be received not later than noon on Saturday. Orders for reprints must be sent to the Journal office, 8 The Fenway.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to The New England Journal of Medicine, 8 The Fenway, Boston, Mass.

THE RECENT ENCEPHALITIS EPIDEMIC IN ST. LOUIS

THE epidemic of encephalitis which occurred in St. Louis and its surrounding territory in August and September of this year was of such unusual character that its chief features should be known by all physicians. There were over six hundred cases, with about one hundred and fifteen deaths. The unique characteristics of the epidemic were many. In the first place, the cases occurred in summer, in contradistinction to the usual period of lethargic encephalitis in the winter-time. There were many cases, moreover, in persons of middle age and over; in previous epidemics almost all the cases were in patients in the first three decades of life. Thirdly, the clinical symptoms were somewhat different from those usually associated with epidemic, or lethargic, encephalitis. There were more signs of irritation of the meninges, with headache, stiffness of the neck and Kernig's sign, gastro-intestinal symptoms were frequently present, and there was a noticeable absence of cranial nerve involvement, particularly of the third cranial nerve, leading to diplopia, which was so constant a finding in the great epidemic of 1917-1919. Pathologically, also, the changes were not confined to the region of the basal ganglia and the brain stem, but were widespread through both the gray and white matter of the cerebrum and cerebellum. These, and one or two other findings, indicate that the St. Louis epidemic consisted of cases differing considerably from von Economo's lethargic type. A group of patients showing somewhat similar symptoms were observed in Japan in 1924 and are now classified as "encephalitis B". It is possible that the Australian "X disease", described a few years ago, was also "encephalitis B".

The high death rate in the St. Louis epidemic was in part due to the age of the patients, many of the older cases having associated arteriosclerosis and hypertension. In those patients that have survived the acute disease, there seems to be no evidence of late manifestations such as have frequently occurred in the ordinary type of epidemic encephalitis. It is too early, however, to make any definite statement in regard to this point, for many cases of encephalitis lethargica, so-called, passed through an acute stage with but transitory symptoms, only to develop marked parkinsonism two or three years subsequently.

The St. Louis epidemic, coming on in such a localized area, has led to a widespread investigation of the epidemiology of the disease. Coöperation between the local, state and federal authorities has been excellent and a considerable amount of research work has been undertaken in order to determine the etiological factor. It is too early to draw any conclusions from the data already presented, except to note that the disease is probably caused by a filterable virus and that it may be transmitted to higher animals by inoculation experiments. The medical world waits with great interest for the final reports on this aspect of the disease, particularly those studies which have been inaugurated by the United States Public Health Service. There seems to be only slight evidence that this particular form of encephalitis has occurred other than in the St. Louis area, although there are a certain number of cases of ordinary epidemic encephalitis appearing sporadically throughout the country.

A JUST RECOGNITION

THE election of Dr. Robert B. Greenough as President-Elect of the American College of Surgeons is an exhibition of appreciation of one who has made an impressive contribution to surgery.

Like many others, Dr. Greenough after having established a reputation as a master in the art

and science of surgery, gave concentrated study to one of its important fields, that of cancer, and has in association with others been a leading factor in standardizing the treatment of this human scourge.

His connection with hospitals, and the State Department of Public Health enabled him to exert great influence in this field.

The profession will always recognize his great contributions which have been singularly devoid of personal exploitation.

As President of the Massachusetts Medical Society his administration demonstrated unusual qualities of leadership consistent with all of his activities.

The profession of New England is justly proud of the advancement of one of its exponents of the traditions of this section, to the Presidency of the American College of Surgeons.

SUFFOLK DISTRICT MEDICAL SOCIETY

In order to obviate the possibility of insufficient appreciation of the fact that Dr. Oliver Hazard Perry Pepper is to be the principal speaker at the first meeting of the Suffolk District Medical Society, the *Journal* urges upon the members of this District to be present that they may extend the courtesy of the District to this eminent representative of the profession in Philadelphia.

Date: October 25. Place: Boston Medical Library. Time: 8:15 P.M.

THIS WEEK'S ISSUE

Contains articles by the following named authors:

Pope, Alton S. A.B., Dr. P.H., M.D. Tufts College Medical School 1924. Formerly Assistant Professor of Epidemiology, College of Physicians and Surgeons, Columbia University. Chief, Bureau of Communicable Diseases, Chicago Department of Health. Now Director, Division of Tuberculosis, Massachusetts Department of Public Health 1930-. His subject is "Pulmonary Tuberculosis in Adolescents with Special Reference to Frequency, Diagnosis, and Prognosis." Page 765. Address: Department of Public Health, 519 State House, Boston.

Holmes, George W. M.D. Tufts College Medical School 1906. Roentgenologist, Massachusetts General Hospital. Clinical Professor, Roentgenology, Harvard Medical School. His subject is "Some Conditions in Which the Roentgen Examination May Lead to an Erroneous Diagnosis of Pulmonary Tuberculosis." Page 774. Address: Massachusetts General Hospital, Boston.

Amberson, J. Burns, Jr. Ph.B., M.D. Johns Hopkins University School of Medicine 1917. Visiting Physician, Bellevue Hospital. Assistant Professor of Clinical Medicine, College of Physicians and Surgeons, Columbia University. His subject is "The Changing Aspect of Tuberculosis Treatment." Page 777. Address: 651 West 238th Street, New York City.

Putnam, Tracy J. M.D. Harvard University Medical School 1920. Assistant Professor of Neurological Surgery and Instructor in Neuropathology, Harvard Medical School. Assistant Visiting Surgeon, Massachusetts General Hospital. His subject is "The Pathogenesis of Multiple Sclerosis: A Possible Vascular Factor." Page 786. Address: Massachusetts General Hospital, Boston, Mass.

Allen, Lyman. A.B., M.Sc., M.D. University of Vermont College of Medicine 1896. F.A.C.S. Professor of Surgery, University of Vermont College of Medicine. His subject is "A Sketch of Vermont's Early Medical History." Page 792. Address: 278 Main Street, Burlington, Vermont.

Little, H. M. B.A., M.D., C.M. McGill University Faculty of Medicine 1901. F.A.C.S. Gynaecologist, Montreal General Hospital. His subject is "Cesarean Section." Page 799. Address: 1374 Sherbrooke Street, West, Montreal.

Patrick, Ivan Y. B.A., M.D., C.M. McGill University Faculty of Medicine 1918, M.C.O.G. Associate in Obstetrics and Gynaecology, Royal Victoria Hospital, Montreal. Demonstrator in Obstetrics and Gynaecology, McGill University. His subject is "Ante Natal Care." Page 801. Address: 524 Argyle Avenue, Westmount, Que.

MASSACHUSETTS GENERAL HOSPITAL

Physicians Invited to Attend Staff Meetings in Medicine, Surgery, Neurology and Pathology

For a number of years clinico-pathological conferences held at the Massachusetts General Hospital have been well attended by physicians of Boston and the Metropolitan District, and the Cabot Case Records, originally analyzed at this hospital and subsequently published in this *Journal* have served many years of useful service.

The hospital wishes to continue to offer physicians access to its unexcelled pathological material, and believes this can best be done at its weekly meeting at noon on Thursdays. At these exercises the clinical case record is first reviewed, x-ray and laboratory data presented, the case discussed by the staff members chiefly concerned, and finally the pathology, both gross and microscopic, is shown. Two or three cases are taken

up in the hour. Needless to say all services of the hospital are represented, and discussion is welcomed.

From time to time there have been requests for clinical meetings. For a number of years the medical and neurological services have held staff meetings of this character, drawing from the wards and out-patient departments patients representing a great variety of problems in diagnosis and treatment. We feel that physicians not on our staff would profit by these presentations. We believe also that the staff members would receive help in solving their problems by a wider discussion than is possible within a limited staff group.

Besides the above meetings which have regularly been held on Thursday mornings, it has been possible to arrange two surgical conferences, also clinical in character, for the same day. These, like the medical exercises will be of diagnostic and therapeutic value to the general practitioner as well as to the specialist. Operations will not be performed at this time.

In these staff meetings it is understood that no attempt at teaching is made, and that they in no way replace or supplant the teaching now carried out in special graduate courses.

For convenience of visitors and staffs, the five meetings are held, according to the following schedule, throughout Thursday morning and will continue during the year except in the summer months. Time and place are as follows:

8:00 A.M.- 9:00 A.M.	Ward rounds. Cases of Peripheral Vascular Disease. Rounds start in Ward I.
9:00 A.M.-10:00 A.M.	Surgical Clinical Conference. Bigelow Amphitheatre.
9:00 A.M.-10:45 A.M.	Neurological Staff Conference. Bulfinch Dome.
11:00 A.M.-12:00 M.	Medical Staff Conference. Bulfinch Dome.
12:00 M.- 1:00 P.M.	Clinico - Pathological Conference. Pathological Amphitheatre.

It is hoped that physicians will feel free to attend any or all of these meetings.

FREDERIC A. WASHBURN, *Director.*

CORRESPONDENCE

OFFICIAL ACTION OF THE BOARD OF REGISTRATION IN MEDICINE

October 3, 1933.

Editor, *New England Journal of Medicine,*

At the meeting of the Board of Registration in Medicine, held September 28, 1933, it was voted to suspend the license of Dr. Dwight F. Willis, 195 Huntington Avenue, Boston, because of his conviction in Court for conspiracy to perform an illegal operation.

STEPHEN RUSHMORE, M.D., *Secretary.*

RECENT DEATHS

HALL—SARAH ADELAIDE HALL, M.D., formerly a practitioner in Watertown, Mass., died in St. Petersburg, Florida, on September 25. Dr. Hall was the widow of Edward C. Hall and a graduate of Tufts College Medical School in 1888. She practiced medicine in Watertown, Mass., for seven years where she was also interested in local philanthropic and health activities. She is survived by a son, Edward C. Hall, who is a member of the Watertown Water Commission.

MEHAN — JOSEPH ALOYSIUS MEHAN, M.D., of Lowell, Massachusetts, died in that city October 12, 1933. He was born in Lowell in 1876 the son of the late Owen and Jane (Flynn) Mehan, and received his early education in his native city and graduated in medicine from the Tufts College Medical School in 1906. He joined the Massachusetts Medical Society in 1906, taught chemistry in the Tufts Medical School for fifteen years and was a member of the staff of St. Johns Hospital where he conducted the roentgenological department. Dr. Mehan was a member of the Council of The Massachusetts Medical Society. He is survived by three children, Frederick L., Mona, and Rosemary Mehan. Two sisters, Mary H., and Jane Mehan, also survive him.

JACKSON — WILLIAM BENJAMIN JACKSON, M.D., of 1640 Cambridge Street, Cambridge, Massachusetts, died in that city October 6, 1933, after an illness of several months. He was born in Dracut in 1853, and after preliminary education in a New Hampshire State Normal School entered the Medical School of Harvard University and graduated in 1880. He devoted his work to surgery in Lowell, Mass., for thirty years, with service on the staffs of the Lowell Corporation and the Lowell General Hospitals. He was at one time chairman of the City Board of Health.

Dr. Jackson joined the Massachusetts Medical Society in 1880, and retired in 1927. He was also a Fellow of the American Medical Association. He is survived by a son, Lawrence M. Jackson, of Belmont.

NOTICE

CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

On Thursday, October 26, in the amphitheatre of the Peter Bent Brigham Hospital at 3:30 P.M.

there will be a clinic conducted by Dr. Henry A. Christian, Hersey Professor of the Theory and Practice of Physic, in the Harvard Medical School, to which physicians are cordially invited.

REPORTS AND NOTICES AND MEETINGS*

AMERICAN COLLEGE OF SURGEONS

OFFICERS ELECTED

At the annual meeting of the American College of Surgeons, the following officers were elected:

President-Elect—Dr. Robert B. Greenough of Boston.

Vice-Presidents—Dr. Charles A. Dukes, Berkeley, California, and Dr. Roscoe B. Graham of Toronto.

Included in the addition to the Board of Governors to serve for three years, are Dr. Charles L. Scudder, Dr. Lincoln Davis, Dr. Frank H. Lahey, Dr. Ernest A. Codman, all of Boston, and Drs. Samuel C. Harvey, New Haven, Conn., Ezra A. Jones of Manchester, New Hampshire, James R. Miller, Hartford, Conn., Herman C. Pitts of Providence, R. I., Eugene B. Sanger, of Bangor, Maine, Martin B. Tinker of Ithaca, New York, and Philemon E. Truesdale of Fall River, Massachusetts.

THE NEISSERIAN MEDICAL SOCIETY OF MASSACHUSETTS

The next meeting of the Neisserian Medical Society of Massachusetts will be held on Wednesday evening, October 25, 1933, at eight fifteen o'clock, in the upper outpatient amphitheatre of the Massachusetts General Hospital (main entrance).

PROGRAM

The Laboratory in the Diagnosis of Gonorrhea (conclusions based on a questionnaire).

The following bacteriologists will participate in the discussion:

Dr. David L. Belding, Massachusetts Memorial Hospitals

Dr. William A. Hinton, State Wassermann Laboratory

Dr. Tracy Mallory, Massachusetts General Hospital

Dr. Monroe J. Schlesinger, Beth Israel Hospital

Miss Edith A. Beckler, State Department of Public Health

Miss Helen H. Gillett, State Department of Public Health

Dr. Hans Zinsser, Harvard Medical School

Dr. S. B. Wolbach, Harvard Medical School

Dr. R. N. Nye, Boston City Hospital

Dr. H. E. MacMahon, Tufts Medical School

Dr. L. D. Tothergill, Children's Hospital

Dr. James Y. Roger, Lowell City Pathologist.

Dr. Raymond H. Goodale, Worcester City Hospital

Physicians, nurses, medical students are cordially invited.

OSCAR F. COX, JR., M.D., *Secretary.*

*NOTE: Essex North District Medical Society affairs are reported on page 805 as follows: (1) Report of October 18 meeting; (2) notice of Censors' Meeting (November 2) and (3) information regarding Postgraduate Instruction Courses in the District.

SUFFOLK DISTRICT MEDICAL SOCIETY

CENSORS' MEETING

The Censors of the Suffolk District Medical Society will meet for the examination of candidates at the Medical Library, No. 8 The Fenway, Thursday, November 2, 1933, at 4:00 o'clock.

Candidates should make personal application to the Secretary, and present their medical diplomas at least one week before the examination.

GEORGE P. REYNOLDS, M.D., *Secretary.*
311 Beacon Street, Boston.

FOUR COUNTY MEDICAL SOCIETY

Come to the Four County Medical Meeting at the Mercy Hospital, Springfield, from 8:30 A.M. to 2 P.M. —October 25, 1933.

PROGRAM

Exhibits—Basement of New Building

8:30 to 10:30 A.M. General Surgery—Third Floor Old Building, Top Floor New Building.

10:30 A.M. Dry Clinic — Nurses' Home. Drs. Naurison, Derby, Rochford, Millman and Ziter.

11 A.M. Papers:

What We Should Tell Patients With an Incurable Disease. Dr. Brace W. Paddock—Pittsfield.

The Mid-Line Tonsil. Dr. Frank A. Millett—Greenfield.

Errors by the General Practitioner in Orthopedics. Dr. R. Nelson Hatt—Springfield.

12:30 P.M. Luncheon—Distinguished Guests: Dr. William H. Robey, Boston, President Massachusetts Medical Society. Dr. Thomas J. O'Brien, Boston, Executive Secretary to the President.

2:15 P.M. Golf.

THE PHYSICIANS ALLIANCE

A regular meeting of the Society will take place at the Whittier Street Health Unit, Roxbury, on Thursday, October 26, 1933, at 8:15 P.M.

Dr. Elliott C. Cutler, Surgeon-in-Chief of the Peter Bent Brigham Hospital, will be guest speaker. The title of his address will be "The Relation of the Physician to the Hospital Out-Patient Department."

The discussion will be opened by Dr. Irving J. Walker and Dr. Hilbert F. Day.

All physicians are cordially invited to attend.

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Am-

phitheatre (Van Dyke Street entrance), Tuesday evening, October 24, at 8:15 o'clock.

Presentation of Cases.

Subject: Experiences with High Temperatures at Boulder City, Nevada, with Some Clinical Applications. By Dr. D. B. Dill and Dr. Arlie V. Bock.

JOHN HOMANS, M.D., *Secretary.*

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

October 19—New England Medical Society will meet in the Evans Auditorium of the Massachusetts Memorial Hospitals, Boston, at 8 P.M. Speaker: Dr. Eugene Pool.

October 19—New England Hospital for Women and Children will hold its regular clinical conference at 8 P.M.

October 20—Boston University School of Medicine, Clinical Meeting at the Boston City Hospital. 11 A.M. to 12 Noon. Speaker: Dr. Eugene Pool.

October 20—The New England Roentgen Ray Society will meet at the Massachusetts Memorial Hospitals at 8 P.M.

October 21—Tri-City Meeting will be held at 10 A.M. at the New York Hospital, 1304 York Avenue.

October 23 - November 3—The 1933 Graduate Fortnight of the New York Academy of Medicine. The theme during the two weeks of intensive study will be Metabolic Disorders. A complete program may be obtained by writing Dr. Frederick P. Reynolds, The New York Academy of Medicine, 2 East 103rd Street, New York City.

October 24—Harvard Medical Society. See page 813.

October 25—The Neisserian Medical Society of Massachusetts. See page 813.

October 25—Four County Medical Society. See page 813.

October 25-30—International Congress on the Scientific and Social Contest Against Cancer will be held at Madrid. For information address Dr. Julio Bejarano, Atocha, 104. Madrid, Spain.

October 26—The Physicians Alliance. See page 813.

October 26—Clinic at the Peter Bent Brigham Hospital. See page 812

October 27—Massachusetts Psychiatric Society will meet at 8:15 P.M. at the Hotel Somerset, Boston.

October 28—The American Board of Dermatology and Syphilology will hold written examinations for candidates in the following cities: New York City; St. Louis, Mo.; Boston, Mass.; Cleveland, Ohio; San Francisco, Calif.; Philadelphia, Pa.; Chicago, Ill. Information may be obtained from the Secretary, Dr. C. Guy Lane, 416 Marlborough Street, Boston, Mass.

November 2—Clinical Meeting at the Faulkner Hospital, 5 P.M.

November 10—William Harvey Society will meet at the Beth Israel Hospital at 8 P.M.

December 15 and 16—The American Board of Dermatology and Syphilology will hold oral examinations in New York City. Information may be obtained from the Secretary, Dr. C. Guy Lane, 416 Marlborough Street, Boston, Mass.

April 16-20, 1934—The American College of Physicians will hold its Eighteenth Annual Clinical Session in Chicago at the Palmer House. For information write Mr. E. R. Loveland, Executive Secretary, 133-135 South 36th Street, Philadelphia, Pa.

July 24-31, 1934—The IVth International Congress of Radiology will be held in Zurich under the presidency of Professor H. R. Schniz. General Secretary Dr. H. E. Walther, Gloriastrasse 14, Zurich.

September 4, 5, 6, 1934—International Union Against Tuberculosis will be held in Warsaw. For particulars address The National Tuberculosis Association, 450 Seventh Avenue, New York, N. Y.

DISTRICT MEDICAL SOCIETIES

ESSEX NORTH DISTRICT MEDICAL SOCIETY

October 20 and 27—Postgraduate Instruction. See page 813.

November 2—Censors' Meeting. See page 813.

E. S. BAGNALL, M.D., Secretary-Treasurer.

FRANKLIN DISTRICT MEDICAL SOCIETY

Meetings will be held on the second Tuesday of November, January, March and May at the Weldon Hotel, Greenfield, at 11 A.M.

CHARLES MOLINE, M.D., Secretary.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

Schedule 1933-1934

Meetings will take place on November 8 at the Bear Hill Club, Stoneham; January 10 at Melrose; March (2nd Wednesday) at Wakefield, and May (2nd Wednesday) at Winchester.

ALLAN R. CUNNINGHAM, Secretary.
76 Church Street, Winchester, Mass.

MIDDLESEX NORTH DISTRICT MEDICAL SOCIETY

Meetings will be held on October 25, January 31, and April 25 (Annual Meeting).

T. A. STAMAS, M.D., Secretary.

MIDDLESEX SOUTH DISTRICT SOCIETY

A meeting of the Middlesex South District will be held October 25, 1933, at 8 P.M., at the Hotel Continental, Cambridge.

Subject: "Function and Workings of the Workmen's Compensation Law."

Speakers: Joseph A. Parks, Chairman of Industrial Accident Board; Dr. Francis D. Donoghue, Medical Adviser to the Industrial Accident Board; Dr. Charles Mongan, Member of the Public Relations Committee.

ALEXANDER A. LEVI, M.D., Secretary.

NORFOLK DISTRICT MEDICAL SOCIETY

October 31—Stated Meeting, Hotel Kenmore, 8 P.M. Dr. H. J. Inglis.

November 28—Roxbury Masonic Temple, 8 P.M. Pneumonia Symposium, in charge of Dr. Frederick T. Lord.

January 28—Tentatively Roxbury Masonic Temple, 8 P.M. Continuance of Symposium on Diseases of the Blood. Dr. Dameshek and Dr. Straus.

February 27—Place, time, subject, and speaker to be announced.

March 27—Place and time to be announced. Dr. Edward A. Edwards and Dr. Henry H. Faxon. Symposium on the Treatment of Varicose Veins.

May—Annual Meeting. Place, time and program to be announced.

FRANK S. CRUICKSHANK, M.D., Secretary.

NORFOLK SOUTH DISTRICT MEDICAL SOCIETY

November 2—12 noon at Norfolk County Hospital. Stated Meeting. Speaker: Dr. Timothy Leary. Subject: Comparative Experimental Arteriosclerosis with Human Coronary Sclerosis.

December 7—12 noon at Quincy City Hospital. Program by the hospital staff.

January 4—12 noon at Norfolk County Hospital. Speaker to be announced.

February 1—12 noon at Norfolk County Hospital. Stated Meeting. Speaker: Dr. Sara Jordan. Subject: Stomach Ulcers.

March 1—12 noon at Quincy City Hospital. Program by the hospital staff.

April 5—12 noon at Norfolk County Hospital. Speaker: Dr. Elliott P. Joslin. Subject: Diabetes.

May 3—12 noon at Norfolk County Hospital. Annual Meeting. Election of Officers.

N. R. PILLSBURY, M.D., Secretary.

SUFFOLK DISTRICT MEDICAL SOCIETY

October 25—The first meeting of the Suffolk District Medical Society will be held on Wednesday, October 25, 1933, at the Boston Medical Library at 8:15 P.M. As a result of the questionnaire the Committee has made an especial effort and been fortunate enough to secure Dr. Oliver Hazard Perry Pepper of Philadelphia as the principal speaker. Dr. Pepper will speak on "Cerebral Hemorrhage and Thrombosis: The Importance of the Differential Diagnosis to Treatment and the Importance of Prevention."

Dr. Jacob H. Swartz of Boston will also address the Society. His subject will be "Fungus Infections of the Skin."

The Committee hopes that all members will attend this important meeting.

Be sure to reserve the date, Wednesday, October 25. Place: Boston Medical Library. Time: 8:15 P.M.

November 2—Censors' meeting. See page 813.

November 29—To be announced.

January 31—With Boston Medical Library and the Trudeau Society. Lawrason Brown, Principal speaker.

March 28—To be announced.

April 25—Annual Meeting.

GEORGE P. REYNOLDS, M.D., Secretary.

WORCESTER DISTRICT MEDICAL SOCIETY

All meetings to be held on Wednesdays as follows:

November 8—Dinner and scientific program at the Grafton State Hospital, North Grafton, Mass.

December 13—Dinner and scientific program at St. Vincent Hospital, Worcester, Mass.

January 10—Dinner and scientific program at the Worcester City Hospital, Worcester, Mass.

February 14—Dinner and scientific program at the Worcester State Hospital, Worcester, Mass.

March 14—Dinner and scientific program at the Memorial Hospital, Worcester, Mass.

April 11—Open date.

May 9—Annual Meeting. Time and place to be announced later.

ERWIN C. MILLER, M.D., Secretary.